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Asthma in Adults and Schoolchildren (Practice Guideline)—The General Practitioner in Asthma Group, the British Association of Accident and Emergency Medicine, the British Paediatric Respiratory Society and the Royal College of Paediatrics and Child Health. *Thorax* 1997;52 Suppl 1:S2-S8.

Conflict of Interest and Financial Disclosure: Judge the Science, Not the Author (Editorial)—Welch SJ. *Chest* 1997;112(4):865-867.

Decline of FEV₁ by Age and Smoking Status: Facts, Figures, and Fallacies (Review)—Kerstjens HA, Rijcken B, Schouten JP, Postma DS. *Thorax* 1997;52(9):820-827.

Diagnosing Cardiovascular and Lung Pathophysiology from Exercise Gas Exchange (Review)—Wasserman K. *Chest* 1997;112(4):1091-1101.

Patient-Assessed Health Outcomes in Chronic Lung Disease: What Are They, How Do They Help Us, and Where Do We Go From Here? (Review)—Curtis JR, Martin DP, Martin TR. *Am J Respir Crit Care Med* 1997;156(4 Pt 1):1032-1039.

Comparison of Outcome Measures for Patients with Chronic Obstructive Pulmonary Disease (COPD) in an Outpatient Setting. Harper R, Brazier JE, Waterhouse JC, Walters SJ, Jones NM, Howard P. *Thorax* 1997;52(10):879.

BACKGROUND: To assist clinicians and researchers in choosing outcome measures for patients with chronic obstructive pulmonary disease (COPD) attending routine outpatient clinics, a comparative assessment was undertaken of 4 questionnaires designed to reflect the patients' perception of their physical and emotional health in terms of their feasibility, validity, reliability, and responsiveness to health change. **METHODS:** Two condition-specific questionnaires, the St George's Respiratory Questionnaire (SGRQ) and Guyatt's Chronic Respiratory Questionnaire (CRQ), and 2 generic questionnaires, the Short Form-36 Health Survey (SF-36) and Euroqol (EQ), were compared for their discriminative and evaluative properties. Spirometric tests and a walking test were also performed. One hundred fifty-six adults who were clinically judged to have COPD and who attended an outpatient chest clinic were assessed at recruitment and 6 and 12 months later. Patients were also asked whether their health had changed since their last assessment. **RESULTS:** Completion rates and consistency between items for dimensions of the SGRQ were lower than for dimensions of the other questionnaires. The distributions of responses were skewed for certain dimensions in all questionnaires except the CRQ. Validity was supported for all instruments insofar as patients' scores were associated with differences in disease severity. The

generic questionnaires better reflected other health problems. All instruments were reliable over time. The condition specific questionnaires were more responsive between baseline and first follow up visit but this difference did not persist. While certain dimensions of the SF-36 were responsive to patient perceived changes, this did not apply to the derived single index of the EQ. The rating scale of the EQ, however, provided a quick and easy indicator of change. **CONCLUSIONS:** Evidence from this study supports the CRQ and the SF-36 as comprehensive outcome measures for patients with longstanding COPD.

Lung Reduction Surgery in Severe Chronic Obstructive Pulmonary Disease (COPD) Decreases Central Drive and Ventilatory Response to CO₂. Celli BR, Montes de Oca M, Mendez R, Stetz J. *Chest* 1997;112(4):902.

BACKGROUND & OBJECTIVES: Lung volume reduction surgery (LVRS) improves ventilatory function in selected patients with severe chronic obstructive pulmonary disease (COPD). The reasons for the observed benefits include the following: increased elastic recoil, improved airflow, and lesser dynamic hyperinflation, and decreased lung volumes. We reasoned that these changes could also alter respiratory drive. **METHODS:** Respiratory central drive was prospectively assessed using the mouth occlusion pressure (P₀₁), and the P₀₁ response to increasing CO₂ (P₀₁/P_{ETCO₂} [end-tidal CO₂ pressure]), in 8 sequential patients before and 3 to 5 months after LVRS. Results were compared with those from 13 control subjects. RE-

SULTS: LVRS decreased total lung capacity from 7.44 ± 1.8 L to 5.92 ± 1.3 L (p < 0.05) and residual volume from 4.97 ± 1.5 L to 3.56 ± 1.1 L (p < 0.05). It also significantly improved FEV₁ from 0.85 ± 0.26 L to 0.99 ± 0.26 L (p < 0.05). Baseline P₀₁ (3.4 ± 1.8 vs 1.4 ± 0.4 cm H₂O, p < 0.01) and P₀₁/P_{ETCO₂} (0.24 ± 0.07 vs 0.11 ± 0.04 cm H₂O/mm Hg, p < 0.05) were higher in patients than in control subjects. After LVRS, P₀₁ decreased from 3.4 ± 1.8 to 1.3 ± 0.75 cm H₂O (p < 0.01) and P₀₁/P_{ETCO₂} from 0.24 ± 0.07 to 0.16 ± 0.06 cm H₂O/mm Hg (p < 0.05). These postoperative values were similar to those of control subjects. There were no correlations between changes in the factors known to influence central drive (P_{aO₂}, P_{aCO₂}, age, weight, height, FVC, and FEV₁) and changes in P₀₁. **CONCLUSIONS:** We conclude that decreased ventilatory drive should be added to the list of benefits of LVRS, and may help explain the symptomatic improvement reported by many patients after this surgery.

Love, Boundaries, and the Patient-Physician Relationship. Farber NJ, Novack DH, O'Brien MK. *Arch Intern Med* 1997;157(20):2291.

Physicians often use their relationships with patients to promote specific therapeutic goals. Because of their personal histories, values, and biases, patients may react to physicians in ways that inhibit or enhance the relationship. The feelings that are aroused may induce physicians to become overly distant, engendering patient and physician dissatisfaction, or to become overly involved emotionally, which can have serious psychological

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and clinical consequences. We explore how a balance between clinical objectivity and bonding with the patient is optimal and achievable. The nature and origin of personal boundaries are described. Boundary transgressions on the part of the patient are discussed, and the means of preventing transgressions by both patients and physicians through medical education, the process of self-awareness, and an exploration of family-of-origin issues are proposed. Through attention to communication with patients, the physician can maintain an empathetic yet objective relationship with the patient.

Stability of Improvements in Exercise Performance and Quality of Life following Bilateral Lung Volume Reduction Surgery in Severe Chronic Obstructive Pulmonary Disease (COPD). Cordova F, O'Brien G, Furukawa S, Kuzma AM, Travaline J, Criner GJ. Chest 1997;112(4):907.

OBJECTIVE: To evaluate the long-term stability of improvements in exercise capacity and quality of life (QOL) after lung volume reduction surgery (LVRS). **DESIGN:** Case-series analysis. **SETTING:** University hospital. **PATIENTS:** Twenty-six patients with severe airflow obstruction (mean FEV₁ of 0.67 ± 0.18 L) and moderate to severe hyperinflation (mean total lung capacity of 7.30 ± 1.90 L). **INTERVENTION & MEASUREMENTS:** All patients underwent bilateral LVRS via median sternotomy. Serial measurement of lung function, symptom-limited cardiopulmonary exercise tests,

6-minute walk distances (6MWD), and sickness impact profile (SIP) scores were done before, and at 3, 6, 12, and 18 months after surgery. **RESULTS:** FEV₁ (0.93 ± 0.29 vs 0.68 ± 0.19 L, p < 0.001) increased while residual volume (3.47 ± 1.2 vs 4.77 ± 1.5 L, p < 0.001) decreased significantly at 3 months post-LVRS compared to baseline, and these changes were maintained at 12 to 18 months follow-up. Similarly, the increase in 6MWD at 3 months post-LVRS (340 ± 84 vs 251 ± 114 m, p < 0.001) was sustained at all follow-up times. On cardiopulmonary exercise testing, total exercise time (9.0 ± 1.8 vs 6.1 ± 1.9 min, p < 0.001), oxygen uptake at peak exercise ($\dot{V}_{O_2}$ peak) (14.9 ± 4 vs 11.9 ± 3 mL/kg/min, p < 0.001), maximum oxygen pulse (7.43 ± 2.37 vs 5.85 ± 1.96 mL/beat, p < 0.005), and maximum minute ventilation ($\dot{V}_{Emax}$) (30.3 ± 10 vs 23.5 ± 7.1 L/min, p < 0.001) increased significantly at 3 months post-LVRS. On serial study following LVRS, total exercise time remained significantly greater at 6 (8.5 ± 1.38 min) and 12 months (8.71 ± 2.0 min) post-LVRS compared to baseline (5.81 ± 1.9 min, p < 0.05). $\dot{V}_{O_2}$ peak tended to be higher at all follow-up periods (3 months, 16.1 ± 4.3; 6 months, 14.5 ± 2.6; 12 months, 14.1 ± 3.5 mL/kg) compared to baseline (12.6 ± 3.9 mL/kg, p = 0.08). Similarly, maximum O₂ pulse tended to be higher in all follow-up studies (3 months, 8.45 ± 2.7; 6 months, 7.6 ± 1.7; 12 months, 7.42 ± 2.1 mL/beat) compared to baseline (6.39 ± 2.5 mL/beat, p = 0.06). Higher $\dot{V}_{Emax}$ continued to be observed at 6 (30 ± 10 L/min) and 12 months (28 ± 10 L/min) post-LVRS, compared to baseline (23 ± 7 L/min,

p = 0.02). $\dot{V}_{Emax}$ post-LVRS was significantly higher at 3 and 6 months compared to baseline on post-hoc analysis (p < 0.05). Overall SIP scores were lower at 3 months (7 vs 18, p < 0.0002) post-LVRS and were sustained in long-term follow-up. **CONCLUSION:** We conclude that bilateral LVRS via median sternotomy in selected patients with severe, diffuse emphysema improves exercise performance and QOL at 3 months following LVRS and these improvements are maintained for at least 12 to 18 months in follow-up.

A Comparison of Sustained-Release Bupropion and Placebo for Smoking Cessation—Hurt RD, Sachs DP, Glover ED, Offord KP, Johnston JA, Dale LC, et al. N Engl J Med 1997;337(17):1195.

BACKGROUND & METHODS: Trials of antidepressant medications for smoking cessation have had mixed results. We conducted a double-blind, placebo-controlled trial of a sustained-release form of bupropion for smoking cessation. We excluded smokers with current depression, but not those with a history of major depression. The 615 subjects were randomly assigned to receive placebo or bupropion at a dose of 100, 150, or 300 mg per day for 7 weeks. The target quitting date (or "target quit date") was 1 week after the beginning of treatment. Brief counseling was provided at base line, weekly during treatment, and at 8, 12, 26, and 52 weeks. Self-reported abstinence was confirmed by a carbon monoxide concentration in expired air of 10 ppm or less. **RESULTS:** At the end of 7 weeks of

treatment, the rates of smoking cessation as confirmed by carbon monoxide measurements were 19.0% in the placebo group, 28.8% in the 100-mg group, 38.6% in the 150-mg group, and 44.2% in the 300-mg group ($p < 0.001$). At one year the respective rates were 12.4%, 19.6%, 22.9%, and 23.1%. The rates for the 150-mg group ($p = 0.02$) and the 300-mg group ($p = 0.01$)—but not the 100-mg group ($p = 0.09$)—were significantly better than those for the placebo group. Among the subjects who were continuously abstinent through the end of treatment, the mean absolute weight gain was inversely associated with the dose (a gain of 2.9 kg in the placebo group, 2.3 kg in 100-mg and 150-mg groups, and 1.5 kg in the 300-mg group; $p = 0.02$). No effects of treatment were observed on depression scores as measured serially by the Beck Depression Inventory. Thirty-seven subjects stopped treatment prematurely because of adverse events; the frequency was similar among all groups. **CONCLUSIONS:** A sustained-release form of bupropion was effective for smoking cessation and was accompanied by reduced weight gain and minimal side effects. Many participants in all groups were smoking at 1 year.

A Predictive Morphometric Model for the Obstructive Sleep Apnea Syndrome—Kushida CA, Efron B, Guilleminault C. *Ann Intern Med* 1997;127(8 Pt 1):581.

BACKGROUND: Mathematical formulas have been used to clinically predict whether patients will develop the obstructive sleep apnea syndrome (OSAS). However, these models do not take into account the disproportionate craniofacial anatomy that accompanies OSAS independently of obesity. **OBJECTIVE:** To determine the accuracy of a morphometric model, which combines measurements of the oral cavity with body mass index and neck circumference, in predicting whether a patient has OSAS. **DESIGN:** 6-month prospective study. **SETTING:** University-based tertiary referral sleep clinic and research center. **PARTICIPANTS:** 300 consecutive patients evaluated for sleep disorders for the first time. **MEASUREMENTS:** Body mass index, neck circumference, and oral cavity measurements were obtained, and a model value was calculated for each patient. Polysomnography was used to determine the number of abnormal respiratory events that occurred during sleep. Sleep apnea was defined as > 5 episodes of apnea or hypopnea/hour of sleep. **RESULTS:** The morphometric model had a sensitivity of 97.6% (95% CI, 95% to 98.9%), a specificity of 100% (CI, 92% to 100%), a positive predictive value of 100% (CI, 98.5% to 100%), and a negative predictive value of 88.5% (CI, 77% to 95%). No significant discrepancies were revealed in tests of intermeasurer and test-retest reliability. **CONCLUSIONS:** The morphometric model provides a rapid, accurate, and reproducible method for predicting whether patients in an ambulatory setting have OSAS. The model may be clinically

useful as a screening tool for OSAS rather than as a replacement for polysomnography.

Efficacy of Automatic Continuous Positive Airway Pressure Therapy that Uses an Estimated Required Pressure in the Treatment of the Obstructive Sleep Apnea Syndrome—Series F, Marc I. *Ann Intern Med* 1997;127(8 Pt 1):588.

BACKGROUND: Continuous positive airway pressure (CPAP) is effective therapy for the obstructive sleep apnea syndrome (OSAS). Automatic CPAP devices continuously adjust the positive pressure to the required levels. **OBJECTIVE:** To determine the efficacy of an automatic CPAP machine used with an estimated reference pressure value. **DESIGN:** A before-and-after, single-blind trial in which patients were randomly allocated to 1 of 3 modes of CPAP administration. **SETTING:** Referral-based sleep center in a public health care institution. **PATIENTS:** 36 outpatients with OSAS. **INTERVENTION:** Continuous positive airway pressure was given at a conventional fixed pressure (Group 1), automatic CPAP was given at a measured reference pressure (Group 2), and automatic CPAP was given at an estimated reference pressure (Group 3). In Group 1, the effective pressure was determined during a titration sleep study. In Groups 2 and 3, the pressure interval was allowed to vary from 4 cm H₂O below reference pressure to 3 cm H₂O above reference pressure. In Group 3, the estimated value of the reference pressure was determined according to individual anthropometric characteristics. **MEASUREMENTS:** Sleep studies were performed and measurements of diurnal sleepiness were obtained at each visit. **RESULTS:** Sleep and breathing disorders and hypersomnolence were alleviated similarly in the 3 groups. The apnea + hypopnea index remained abnormal in 1 patient in Group 3 for whom the reference pressure had been underestimated. A strong negative correlation was found between the percentage of time spent below reference pressure during CPAP and the difference between the effective and estimated pressures. **CONCLUSION:** Automatic CPAP can be used with an estimated reference pressure without doing a titration sleep study. The positive pressure trend can be used to determine whether treatment failure is caused by an inadequate pressure setting and to determine the amount of pressure to apply.

A Comparison of Low-Dose Inhaled Budesonide plus Theophylline and High-Dose Inhaled Budesonide for Moderate Asthma. Evans DJ, Taylor DA, Zetterstrom O, Chung KF, O'Connor BJ, Barnes PJ. *N Engl J Med* 1997;337(20):1412.

BACKGROUND: Inhaled glucocorticoids and oral theophylline are widely used to treat asthma. We compared the benefits of adding theophylline to inhaled glucocorticoid with those of doubling the dose of inhaled glucocorticoid in patients with per-

sistent symptoms despite the use of inhaled glucocorticoid. **METHODS:** In a double-blind, placebo-controlled trial, we randomly assigned 62 patients to receive either 400 μ g of inhaled budesonide (low-dose budesonide) with 250 or 375 mg of theophylline (depending on body weight) or 800 μ g of inhaled budesonide (high-dose budesonide). All doses were given twice daily for 3 months. Lung function was measured serially, and patients kept records of peak expiratory flow, symptoms, and albuterol use. The effects of treatment on endogenous cortisol levels were also assessed. **RESULTS:** Both treatments resulted in improvements in lung function that were sustained throughout the study. As compared with treatment with high-dose budesonide, treatment with low-dose budesonide plus theophylline resulted in greater improvements in forced vital capacity ($p = 0.03$) and forced expiratory volume in one second ($p = 0.03$). There were significant and similar reductions in β_2 -agonist use and the variability of peak expiratory flow, a correlate of bronchial hyperresponsiveness and the severity of asthma. Serum cortisol concentrations were significantly reduced in the group given high-dose budesonide (from a mean [$\pm$ standard error]) of 18.4 ± 2.4 μ g/dL to 15.9 ± 2.1 μ g/dL, $p = 0.02$) but were unchanged in the other group. The median serum theophylline concentration was 8.7 μ g per milliliter (therapeutic range, 10 to 20) among those who received theophylline. Both treatments were well tolerated. **CONCLUSIONS:** For patients with moderate asthma and persistent symptoms, low-dose inhaled budesonide with theophylline and high-dose inhaled budesonide produced similar benefits. Effects were achieved at theophylline concentrations below the recommended therapeutic range. The addition of low-dose theophylline to inhaled glucocorticoid may be preferable to and cheaper than increasing the dose of inhaled glucocorticoid.

An Unusual Cause of Cyanosis in a Patient with a Carcinoid Tumor—Rodríguez-Cintrón W, González JM, Zoghbi W, Stevens PM. *South Med J* 1997;90(10):1029.

We report the second case of a right-to-left interatrial shunt due to a carcinoid tumor, which was successfully corrected by surgery. The patient had closure of a patent foramen ovale and tricuspid valve replacement. One and one half years after surgery, the patient had no symptoms related to heart disease.

Cost-Effectiveness of Vaccination Against Pneumococcal Bacteremia among Elderly People—Sisk JE, Moskowitz AJ, Whang W, Lin JD, Fedson DS, McBean AM, et al. *JAMA* 1997;278(16):1333.

CONTEXT: Clinical, epidemiologic, and policy considerations support updating the cost-effectiveness of pneumococcal vaccination for elderly people and targeting the evaluation only to pre-

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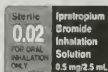
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CONTRAINDICATIONS: Ipratropium bromide is contraindicated in known or suspected cases of hypersensitivity to ipratropium bromide, or to atropine and its derivatives.

WARNINGS: The use of Ipratropium Bromide Inhalation Solution as a single agent for the relief of bronchospasm in acute COPD exacerbation has not been adequately studied. Drugs with faster onset of action may be preferable as initial therapy in this situation. Combination of Ipratropium Bromide Inhalation Solution and beta agonists has not been shown to be more effective than either drug alone in reversing the bronchospasm associated with acute COPD exacerbation.

Immediate hypersensitivity reactions may occur after administration of ipratropium bromide, as demonstrated by rare cases of urticaria, angioedema, rash, bronchospasm and oropharyngeal edema.

PRECAUTIONS: General: Ipratropium bromide should be used with caution in patients with narrow angle glaucoma, prostatic hypertrophy or bladder neck obstruction.

Information for Patients: Patients should be advised that temporary blurring of vision, precipitation or worsening of narrow-angle glaucoma or eye pain may result if the solution comes into direct contact with the eyes. Use of a nebulizer with mouthpiece rather than face mask may be preferable, to reduce the likelihood of the nebulizer solution reaching the eyes. Patients should be advised that Ipratropium Bromide Inhalation Solution can be mixed in the nebulizer with albuterol or metaproterenol if used within one hour. Drug stability and safety of Ipratropium Bromide Inhalation Solution when mixed with other drugs in a nebulizer have not been established. Patients should be reminded that Ipratropium Bromide Inhalation Solution should be used consistently as prescribed throughout the course of therapy.

Drug Interactions: Ipratropium bromide has been shown to be a safe and effective bronchodilator when used in conjunction with beta adrenergic bronchodilators. Ipratropium bromide has also been used with other pulmonary medications, including methylxanthines and corticosteroids, without adverse drug interactions.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Two-year oral carcinogenicity studies in rats and mice have revealed no carcinogenic potential at dietary doses up to 6 mg/kg/day of ipratropium bromide.

Results of various mutagenicity studies (Ames test, mouse dominant lethal test, mouse micronucleus test and chromosome aberration of bone marrow in Chinese hamsters) were negative.

Fertility of male or female rats at oral doses up to 50 mg/kg/day was unaffected by ipratropium bromide administration. At doses above 90 mg/kg, increased resorption and decreased conception rates were observed.

Pregnancy: TERATOGENIC EFFECTS:

Pregnancy Category B. Oral reproduction studies in mice, rats and rabbits at doses of 10, 100, and 125 mg/kg respectively and inhalation reproduction studies in rats and rabbits at doses of 1.5 and 1.8 mg/kg (or approximately 38 and 45 times the recommended human daily dose) respectively, have demonstrated no evidence of teratogenic effects as a result of ipratropium bromide. However, no adequate or well-controlled studies have been conducted in pregnant women. Because animal reproduction studies are not always predictive of human response, ipratropium bromide should be used during pregnancy only if clearly needed.

Nursing Mothers: It is not known whether ipratropium bromide is excreted in human milk. Although lipid-insoluble gustatory bases pass into breast milk, it is unlikely that ipratropium bromide would reach the infant to a significant extent, especially when taken by inhalation since ipratropium bromide is not well absorbed systemically after inhalation or oral administration. However, because many drugs are excreted in human milk, caution should be exercised when ipratropium bromide is administered to a nursing woman.

Pediatric Use: Safety and effectiveness in pediatric patients below the age of 12 have not been established.

ADVERSE REACTIONS: Adverse reaction information concerning Ipratropium Bromide Inhalation Solution is derived from 12-week active-controlled clinical trials. Additional information is derived from foreign post-marketing experience and the published literature.

All adverse events, regardless of drug relationship, reported by three percent or more patients in the 12-week controlled clinical trials appear in the table.

All Adverse Events, From a Double-blind, Parallel, 12-week Study of Patients with COPD*

	PERCENT OF PATIENTS				
	Ipratropium Bromide (500 mcg t.i.d.) n = 219	Meta- proterenol (15 mg t.i.d.) n = 212	Ipratropium / Bromide / Metaproterenol (500 mcg t.i.d. / 15 mg t.i.d.) n = 108	Albuterol (2.5 mg t.i.d.) n = 205	Ipratropium Bromide / Albuterol (500 mcg t.i.d. / 2.5 mg t.i.d.) n = 100
Body as a Whole:					
General Disorders					
Headache	6.4	5.2	6.5	6.3	9.0
Pain	4.1	3.3	0.9	2.9	5.0
Influenza-like symptoms	3.7	4.7	6.5	0.5	1.0
Back Pain	3.2	1.9	1.9	2.4	0.0
Chest Pain	3.2	4.2	5.6	2.0	1.0
Cardiovascular Disorders					
Hypertension/ Hypertension Aggravated	0.9	1.9	0.9	1.5	4.0
Central & Peripheral Nervous System					
Dizziness	2.3	3.3	1.9	3.9	4.0
Insomnia	0.9	0.5	4.6	1.0	1.0
Tremor	0.9	7.1	8.3	1.0	0.0
Nervousness	0.5	4.7	6.5	1.0	1.0
Gastrointestinal System Disorders					
Mouth Dryness	3.2	0.0	1.9	2.0	3.0
Nausea	4.1	3.8	1.9	2.9	2.0
Constipation	0.9	0.0	3.7	1.0	1.0
Musculo-skeletal System Disorders					
Arthritis	0.9	1.4	0.9	0.5	3.0
Respiratory System Disorders (Lower)					
Coughing	4.6	8.0	6.5	5.4	6.0
Dyspnea	9.6	13.2	16.7	12.7	9.0
Bronchitis	14.6	24.5	15.7	16.6	20.0
Bronchospasm	2.3	2.8	4.6	5.4	5.0
Sputum Increased	1.4	1.4	4.6	3.4	0.0
Respiratory System Disorders (Upper)					
Upper Respiratory Tract Infection	13.2	11.3	9.3	12.2	16.0
Pharyngitis	3.7	4.2	5.6	2.9	4.0
Rhinitis	2.3	4.2	1.9	2.4	0.0
Sinusitis	2.3	2.8	0.9	5.4	4.0

*All adverse events, regardless of drug relationship, reported by the three percent or more patients in the 12-week controlled clinical trials

Additional adverse reactions reported in less than three percent of the patients treated with ipratropium bromide include tachycardia, palpitations, eye pain, urinary retention, urinary tract infection and urticaria. Cases of precipitation or worsening of narrow-angle glaucoma and acute eye pain have been reported.

Lower respiratory adverse reactions (bronchitis, dyspnea and bronchospasm) were the most common events leading to discontinuation of ipratropium bromide therapy in the 12-week trials. Headache, mouth dryness and aggravation of COPD symptoms are more common when the total daily dose of ipratropium bromide equals or exceeds 2,000 mcg.

Allergic-type reactions such as skin rash, angioedema of tongue, lips and face, urticaria, laryngospasm and anaphylactic reaction have been reported. Many of the patients had a history of allergies to other drugs and/or foods.

HOW SUPPLIED: Ipratropium Bromide Inhalation Solution is a clear, colorless solution supplied in a unit-dose vial containing 2.5 mL. Supplied in cartons as listed below:

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vention of pneumococcal bacteremia. **OBJECTIVE:** To assess the implications for medical costs and health effects of vaccination against pneumococcal bacteremia in elderly people. **DESIGN:** Cost-effectiveness analysis of pneumococcal vaccination compared with no vaccination, from a societal perspective. **SETTING & PARTICIPANTS:** The elderly population aged 65 years and older in the United States in 3 geographic areas: metropolitan Atlanta GA; Franklin County OH; and Monroe County NY. **MAIN OUTCOME MEASURES:** Incremental medical costs and health effects, expressed in quality-adjusted life-years per person vaccinated. **RESULTS:** Vaccination was cost saving, ie, it both reduced medical expenses and improved health, for all age groups and geographic areas analyzed in the base case. For people aged 65 years and older, vaccination saved \$8.27 and gained 1.21 quality-adjusted days of life per person vaccinated. Vaccination of the 23 million elderly people unvaccinated in 1993 would have gained about 78,000 years of healthy life and saved \$194 million. In univariate sensitivity analysis, the results remained cost saving except for doubling vaccination costs, including future medical costs of survivors, and lowering vaccination effectiveness. With assumptions most unfavorable to vaccination, cost per quality-adjusted life-year ranged from \$35 822 for ages 65 to 74 years to

\$598 487 for ages 85 years and older. In probabilistic sensitivity analysis, probability intervals were more narrow, with less than 5% probability that the ratio for ages 85 years and older would exceed \$100,000. **CONCLUSIONS:** Pneumococcal vaccination saves costs in the prevention of bacteremia alone and is greatly underused among the elderly population, on both health and economic grounds. These results support recent recommendations of the Advisory Committee on Immunization Practices and public and private efforts under way to improve vaccination rates.

Effects of Aerosolized Surfactant in Patients with Stable Chronic Bronchitis: A Prospective Randomized Controlled Trial. Anzueto A, Jubran A, Ohar JA, Piquette CA, Rennard SI, Colice G, et al. JAMA 1997;278(17):1426.

CONTEXT: Chronic bronchitis, estimated to affect more than 13 million adults in the United States, is characterized in part by retention of airway secretions, but no approved or effective therapy for airway mucus retention in patients with chronic bronchitis has been established. Surfactant reduces sputum adhesiveness, which contributes to difficulty in clearing secretions, but surfactant has not been tested in patients with chronic bronchitis. **OBJECTIVE:** To examine the effects of exogenous surfactant on sputum clearance and

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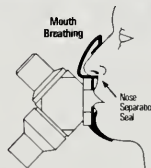
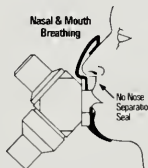
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pulmonary function in patients with stable chronic bronchitis. **DESIGN:** A prospective, multicenter, randomized, double-blind, parallel-group, placebo-controlled comparison of the effects of 2 weeks of treatment with 3 doses of aerosolized surfactant (palmitoylphosphatidylcholine [DPPC]) or saline (placebo). **SETTING:** Four U.S. teaching hospitals. **PARTICIPANTS:** A total of 87 adult patients with the diagnosis of stable chronic bronchitis. **MAIN OUTCOME MEASURES:** Pulmonary function, respiratory symptoms, and sputum properties before treatment (Day 0), after 2 weeks of treatment (Day 14), and 7 days after stopping treatment (Day 21). **RESULTS:** A total of 66 patients were randomized to surfactant treatment and 21 to saline treatment. Patient demographic characteristics between groups were similar at baseline. In patients who received a DPPC dose of 607.5 mg/d for 2 weeks, prebronchodilator forced expiratory volume in 1 second (FEV₁) increased from 1.22 L (standard error of the mean [SEM], 0.08 L) at Day 0 to 1.33 L (SEM, 0.09 L) at Day 21 (p = 0.05), an improvement of 11.4%; postbronchodilator FEV₁ improved 10.4% by Days 14 and 21 (p = 0.02); and the ratio of residual volume to total lung capacity, a measure of thoracic gas trapping, decreased 6.2% by Day 21 (p = 0.009). In the surfactant groups, there was a dose-dependent increase in the ability of sputum to be transported by cilia in vitro. **CONCLU-**

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AARC Assessment Course/Examination Detailed Content Outline

1. Obligations of the Respiratory Care Practitioner in the Practice of Patient Assessment

- A. Appreciate the cost of care
- B. Understand the scope and limitations of the practitioners role in patient assessment
- C. Understand the principle of patient self-determination
- D. Appreciate the psychological, social, and physical characteristics of wellness/illness
- E. Recognize the patient's right to confidentiality
- F. List the principles of informed consent

▶ "Assessment of the patient isn't purely clinical anymore — helped to start the 'Thinking outside the box concept,' that is necessary for continued survival of RT as a profession."

2. Perform a Comprehensive Health Assessment

- A. Review patient records
- B. Structure an interview
- C. Conduct an interview
- D. Conduct a physical assessment
- E. Identify needs for referral
- F. Identify patients with potential for high-risk medical complications

- G. Accurately document assessment findings
- H. Present findings to a physician

▶ "I truly believe that assessment (total — not just physical) is what will keep our profession a vital portion of the health care team."

3. Assess the Caregivers

- A. Identify sites of care and caregivers
- B. Interview the caregivers
- C. Assess the abilities of caregivers to manage physical, technological, and emergency situations
- D. Assess home health resources

4. Assess the Patient's Understanding and Compliance with Physician's Orders and Care Plan

- A. Review the physician's order with the patient
- B. Assess the patient's understanding of technique and components

▶ "I plan on sending other staff who will be involved in asthma case management to the April '98 course in Dallas"

John Kimble, RT
Director, Respiratory Care Services and Sleep Disorders Laboratory Scott & White Memorial Hospital Temple, TX

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▶ "I traveled ten hours to get here and feel it was well worth the trip. Your program is great."

Penny Gagne Plouff, RRT

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- C. Assess patient ability to administer treatments, use equipment, monitor changes, interpret and respond to those changes

- D. Document assessment findings

5. Assess the Patient's Environment as It Relates to Health Status and Supporting Physician's Orders

- A. Identify barriers to wellness

▶ "The course was relevant to the changes in health care and my practice. Well worth my time."

- B. Inspect the patient care environment

- C. Determine the resources available to the patient and family

- D. Document assessment findings

▶ "Bravo for a job well done. I plan to share a lot of this information with staff."

6. Assess Continued Appropriateness of Physician Orders and Care Plan

- A. Review physician orders

- B. Evaluate the patient's response to physician orders

- C. Evaluate the appropriateness of the orders and the care plan

- D. Recommend modification of the treatment plan

- E. Document assessment findings

Course Registration Dallas, TX — Registration Deadline March 30, 1998

* Registration will close on the date noted or when 200 registrations are received.

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For more information about the alliance between the NLHEP & the ACCP, look for the resource document on Page 185 of this issue.

KNOW YOUR NUMBERS

SION: Aerosolized surfactant improved pulmonary function and resulted in a dose-related improvement in sputum transport by cilia in patients with stable chronic bronchitis.

Mycobacteremia in Patients with the Acquired Immunodeficiency Syndrome. Grinsztajn B, Fandinho FC, Veloso VG, Joao EC, Lourenco MC, Nogueira SA, et al. Arch Intern Med 1997; 157(20):2359.

BACKGROUND: Bacilleemia is a key event in the pathogenesis of tuberculosis. Although current evidence indicates that *Mycobacterium tuberculosis* bacteremia is rare in patients seronegative for the human immunodeficiency virus, it has been increasingly reported in patients with the acquired immunodeficiency syndrome (AIDS). **OBJECTIVE:** To determine clinical and laboratory characteristics of patients with AIDS and tuberculosis with and without bacilleemia. **METHODS:** Fifty patients with AIDS with clinical suspicion of disseminated mycobacterial disease were prospectively selected. Three consecutive blood samples were collected for culture using a standardized protocol. **RESULTS:** Mycobacterium was isolated from any body site in 42 patients (84%). Bacilleemia was detected in 30 (71.4%) of these 42 patients: 11 (28.2%) caused by *Mycobacterium avium*-intracellular complex and 19 (71.8%) caused by *M. tuberculosis*. Blood culture was the only method used to confirm the diagnosis in 5 (15%) of the 33 tuberculosis cases. Tuberculosis in patients with AIDS developed with nonspecific insidious symptoms, a remarkable elevated alkaline phosphatase level, and without the classic military radiological pattern. We could demonstrate 2 previously unrevealed clinical characteristics of bacteremic tuberculosis in patients

with AIDS: a shift to the left in the white blood cell count and abdominal lymph node enlargement. In patients with tuberculosis, the in-hospital mortality rate was higher among patients with bacilleemia, although the posttreatment survival rate was comparable. **CONCLUSIONS:** Blood culture is a valuable tool to confirm the clinical diagnosis of disseminated tuberculosis in patients with AIDS and can distinguish patients with characteristic clinical findings and outcome. Abdominal ultrasonography may be an additional helpful tool to identify these patients.

Deciding Life and Death in the Courtroom. From Quinlan to Cruzan, Glucksberg, and Vacco—A Brief History and Analysis of Constitutional Protection of the 'Right to Die'. Gostin LO. JAMA 1997;278(18):1523.

This article analyzes judicial determinations on the "right to die" from Quinlan to Cruzan, Glucksberg, and Vacco. The body of law known as right-to-die cases extends ordinary treatment refusal doctrine to end-of-life decisions. The courts, having affirmed a right to refuse life-sustaining treatment, held that certain categorical distinctions that had been drawn lacked a rational basis. No rational distinction could be made between competent versus incompetent patients, withholding versus withdrawing treatment, and ordinary versus extraordinary treatment. The courts, however, had persistently affirmed one categorical distinction: between withdrawing life-sustaining treatment on the one hand and active euthanasia or physician-assisted dying on the other. In Washington versus Glucksberg and Vacco versus Quill, the Supreme Court unanimously held that physician-assisted suicide is not a fundamental liberty interest pro-

ected by the Constitution. Notably, 5 members of the Court wrote or joined in concurring opinions that took a more liberal view. The Court powerfully approved aggressive palliation of pain. The Supreme Court, hinting that it would find state legalization of physician-assisted suicide constitutional, invited the nation to pursue an earnest debate on physician assistance in the dying process.

Does This Patient Have Community-Acquired Pneumonia? Diagnosing Pneumonia by History and Physical Examination. Metlay JP, Kapoor WN, Fine MJ. JAMA 1997;278(17):1440.

Community-acquired pneumonia is an important cause of acute respiratory symptoms (eg, cough) in the ambulatory care setting. Distinguishing pneumonia from other causes of respiratory illnesses, such as acute bronchitis and upper respiratory tract infections, has important therapeutic and prognostic implications. The reference standard for diagnosing pneumonia is chest radiography, but it is likely that many physicians rely on the patient's history and their physical examination to diagnose or exclude this disease. A review of published studies of patients suspected of having pneumonia reveals that there are no individual clinical findings, or combinations of findings, that can rule in the diagnosis of pneumonia for a patient suspected of having this illness. However, some studies have shown that the absence of any vital sign abnormalities or any abnormalities on chest auscultation substantially reduces the likelihood of pneumonia to a point where further diagnostic evaluation may be unnecessary. This article reviews the literature on the appropriate use of the history and physical examination in diagnosing community-acquired pneumonia.



WATCH FOR A SPECIAL APRIL 1998
RESPIRATORY CARE

SLEEP-DISORDERED
BREATHING

Effects of a Pulmonary Rehabilitation Program on Physiologic Measures, Quality of Life, and Resource Utilization in a Health Maintenance Organization Setting

Louann Parker MT(ASCP) MBA and Judy Walker RRT

INTRODUCTION: This study was designed to examine the physiologic, quality of life (QOL), and resource utilization measures of chronic obstructive pulmonary disease (COPD) patients in our health maintenance organization (HMO) pulmonary rehabilitation program. **METHODS:** We studied 47 COPD patients, aged 54-88 years, who participated in a 7-week pulmonary rehabilitation program from March 1995 through 1996. Patients were selected to participate if they had a diagnosis of COPD (including emphysema, chronic bronchitis, bronchiectasis, and chronic asthma), were symptomatic with impairment in activities of daily living, were motivated to complete the program, had a forced expiratory volume in the first second (FEV₁) of < 60% predicted, had the mental and physical capability to participate, and were medically stable. Patients with heart or neurological conditions were excluded from the study. Patients attended a 7-week core program. Baseline QOL indicators, measured by a survey tool, and 5 pathophysiologic indicators: metabolic equivalents (MET), pulse oximetry saturation (S_{pO₂}), resting heart rate (RHR), functional aerobic impairment (FAI), and walking distance were measured immediately before and after the 7-week program. Charge data for the 12 months pre- and postprogram were retrieved from the HMO's billing system for hospital visits for which the primary diagnosis was COPD. Following the program patients chose whether to attend the Lung Gym, an exercise maintenance program. **RESULTS:** For 6 of the 7 survey questions QOL improved significantly ($p < 0.01$). For the pathologic indicators: MET increased from 2.49 equivalents to 2.72 equivalents ($p < 0.02$); S_{pO₂} increased from 86.7% to 89.9% ($p < 0.01$); RHR decreased from 89 to 84 beats/min ($p < 0.05$); FAI decreased from 59% to 56% ($p < 0.2$) but remained in the marked impairment range; and walking distance was not significantly improved. Utilization costs postprogram decreased to \$939/patient ($p < 0.01$) with the average outpatient charge decreasing from \$82.41 preprogram to \$63.33 postprogram. Both Lung Gym and Non-Lung Gym groups decreased postprogram charges ($p < 0.01$ Lung Gym; $p < 0.05$ Non-Lung Gym). **CONCLUSIONS:** This study revealed significant utilization savings when comparing COPD charge data for the 12-month period before and after completing the 7-week core program. Enhanced quality of life and pathophysiologic improvement were demonstrated using patient perceptions and clinical testing before and after the program. [Respir Care 1998;43(3):177-182]

Introduction

Pulmonary rehabilitation can be clinically effective, as other studies¹⁻⁴ have shown, but no one has examined the efficacy and impact on resource utilization in a health maintenance organization (HMO) setting. The program we report on was

designed to improve the health of chronic obstructive pulmonary disease (COPD) patients. However, because our hospital, clinics, and the HMO are all owned by the same parent company, periodic documentation of patient outcomes and justification of the expenses are required to continue the program. Thus, the tracking of the pathophysiologic and quality of life indicators were an integral part of the program from the beginning.

Improved patient health alone was not enough to justify the program due to health care's continuing efforts to decrease medical costs and maintain the ability to offer competitive premium pricing. Because this organization serves both fee for service (FFS), or traditional indemnity insurance, and HMO

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patients, the focus for remaining competitive in today's health care market has been on increasing revenue from FFS patients, a short-term financial strategy. This program is profitable in the short term because it generates FFS revenue and in the long term because it decreases the utilization of health care services by the HMO patients. This study was used to illustrate the long-term profitability of a pulmonary rehabilitation program serving HMO patients.

Methods

This pulmonary rehabilitation program was established in March 1995 to help outpatient pulmonary patients achieve the highest possible level of independent function through a combination of education and physical conditioning. The design of the program was typical of the majority of such programs around the United States¹ except that it served primarily HMO patients. These patients, clients of a 150,000-member HMO, were referred to the program by the pulmonary rehabilitation medical director, who assessed the patients in the pulmonology clinic.

All patients were referred for pulmonary assessment by their primary care physician, had a diagnosis of COPD (which included emphysema, chronic bronchitis, bronchiectasis, and chronic asthma); were symptomatic due to their lung disease with impairment in activities of daily living; were motivated to complete the program; had a forced expiratory volume in the first second of expiration (FEV₁) of less than 60% of predicted or an FEV₁ less than 1.5 L; had the mental and physical capability to actively participate in the program; and were medically stable. Patients with a myocardial infarction within the previous 6 months, uncontrolled heart failure or arrhythmias, ongoing alcohol or drug abuse, organic brain syndrome or other neurologic problem that would interfere with comprehension, lung cancer with < 1 year prognosis, inadequate motivation to comply with the program, history of prior non-compliance with health care, morbid obesity or profound cachexia which would preclude active exercise, or other serious medical problems were excluded from the program. (No data are available on the number of patients excluded from the program.) Of the 67 patients who began the program during the study period March 1995 through March 1996, 7 (10.5%) dropped out. Another 13 patients were excluded from the study for lack of utilization data because they were FFS patients, were not continuously enrolled in the HMO during the study period, or had expired.

The core program consisted of 3 classes per week for 7 weeks. During each 3-hour class, the instructors used a combination of educational materials, lectures, videos, and group and individual exercise training. This comprehensive design was chosen because it has been well documented that education alone does not provide clinical or functional benefit to COPD patients.²⁻⁴ The core program was supplemented by an optional exercise maintenance program, the 'Lung Gym,'

that some patients (38%) attended 3 times/week.

Only those patients who completed the core program in its entirety were included in this study. The patients ranged in age from 54-88 years with a median age of 75 (mean = 73). At the start of the program, the average functional aerobic impairment (FAI)^{5,6} was 59% which is defined as indicating "marked impairment." Of the 47 patients included in the study, 18 of them chose to continue their rehabilitation through attendance at the Lung Gym. At the beginning of the program, a baseline was established for each patient for quality of life (QOL) and 5 pathophysiologic indicators. These measures were repeated upon completion of the core program to determine whether any significant subjective and objective improvement had occurred.

A short, 7-question QOL survey was used to assess patient perceptions of their shortness of breath during exercise, understanding of their lung disease and medications, ability to exercise, cope with their illness and perform daily activities, and overall QOL on a 1 (poor) to 10 (very good) scale. The questionnaire was internally developed as a much shorter version of the previously validated lengthy questionnaires, such as St George's Respiratory Questionnaire,⁷ Chronic Respiratory Disease Questionnaire,⁸ and Pulmonary Functional Status and Dyspnea Questionnaire,⁹ covering similar topics in greater detail. The questionnaire was self administered, and the patients were allowed access to their preprogram scores while they completed the questionnaire at the end of the program. In conjunction with a 6-minute walk test, the pathophysiologic indicators chosen for this study were metabolic equivalents (MET), pulse oximetry saturation (SpO₂),* resting heart rate (RHR), FAI, and walking distance. Standard testing protocols were used as described in texts about exercise testing.¹⁰⁻¹²

The resource utilization data were collected retrospectively, 2 years after the program began, to further support continuation of the program. Charge data for the 47 patients in the study were retrieved from the HMO's computerized billing system for outpatient visits including laboratory testing, hospital admissions, emergency department visits, and home care visits where the primary diagnosis was COPD. The data were analyzed to determine average charges and number of visits for the group as a whole, for the Lung Gym participants group and for the Non-Lung Gym group (ie, the 29 patients who chose not to participate in the exercise maintenance component of the program) for the 12-month period prior to entering the program (preprogram) and the 12-month period after completing the program (postprogram). Data for the 7 weeks during which the patients were in the core program and the Lung Gym visits were excluded from the analysis.

Two-tailed *t* tests at an α level of 0.05 were used to test for differences between the preprogram and postprogram annual charges/patient as well as the QOL and physiologic indicators. The results were evaluated for the patients as a whole and for the Lung Gym group and the Non-Lung Gym group.

Results

Figure 1 shows that the patients' perceptions of their QOL improved significantly. For 6 of the 7 questions, including a decreased shortness of breath during exertion, an increased ability to exercise and perform daily activities, and an increased understanding of their disease and medications ($p < 0.01$), QOL improved significantly. The patients reported an insignificant increase in their ability to cope with their disease ($p < 0.1$).

The subjective findings were consistent with the fact that 3 of the 5 objective pathophysiologic indicators showed improvement from the baseline (Fig. 2). MET increased significantly ($p < 0.02$) from 2.49 equivalents at the beginning of the core program to 2.72 equivalents at the end. S_{pO_2} increased significantly ($p < 0.01$) from 86.7% to 89.9%, while RHR improved (decreased) significantly ($p < 0.05$) from 89 to 84 beats/min. FAI improved (decreased) insignificantly ($p < 0.2$) from 59% to 56% but remained in the "marked impairment" range of 55-68% after the 7 weeks of rehabilitation. Walking distance also improved (increased) insignificantly ($p < 0.1$) from 1,147 ft to 1,274 ft.

For the patients as a whole, the postprogram charges were significantly decreased ($p < 0.01$) when compared to the preprogram charges, resulting in average decreased utilization charges of \$939/patient for the 12 months following completion

of the program, a combined effect of significantly decreased charges/visit ($p < 0.01$) and an insignificant average decrease of 2.5 charges/year (Fig. 3). Of the charges billed for these patients, 95% were related to outpatient visits and testing. The average outpatient charge decreased from \$82.41 preprogram to \$63.33 postprogram ($p < 0.01$).

Both the Lung Gym group and the Non-Lung Gym group also had significantly decreased postprogram charges ($p < 0.01$, Lung Gym; $p < 0.05$ for Non-Lung Gym). The Lung Gym group (Fig. 4A) realized a decrease of \$778/per patient while the Non-Lung Gym group (Fig. 4B) had a decrease of \$1,038/patient. Both groups experienced a 55% decrease in charges/patient per year because the Lung Gym group had lower preprogram charges than the Non-Lung Gym group.

Discussion

We believe this program is an excellent example of the kinds of activities health care organizations should be focusing on: investment in disease specific patient education/rehabilitation programs that have been proven to benefit both the patients and the organization. The benefits may take more than 1 budget cycle to realize, but they are especially valuable precisely because of their long-term nature.

The findings in this study support many of the conclusions reached at a 1994 National Institute of Health Pulmonary Reha-

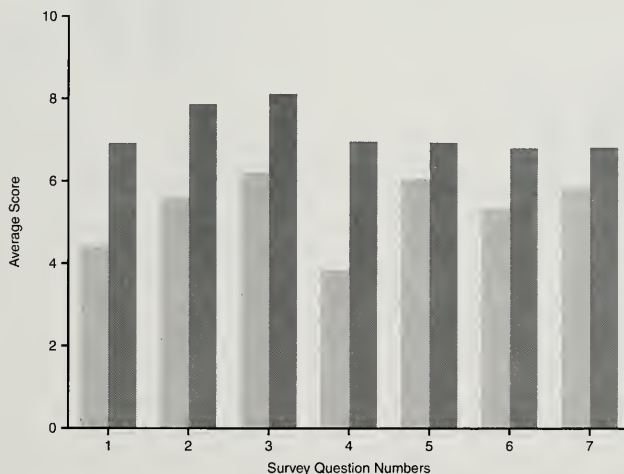


Fig. 1. The results of the Quality of Life survey administered to patients at the beginning and upon completion of the core 7-week program. Rated on a scale of 1 = poor to 10 = very good, the fill-in-the-blank survey questions were: 1 = In general, my shortness of breath during exertion is; 2 = My understanding of my lung disease is; 3 = My understanding of my medications is; 4 = In general, my ability to exercise is; 5 = My ability to cope with my illness and stress is; 6 = My ability to do my daily activities is; and 7 = In general, my quality of life is. ■ = preprogram; ■ = postprogram.

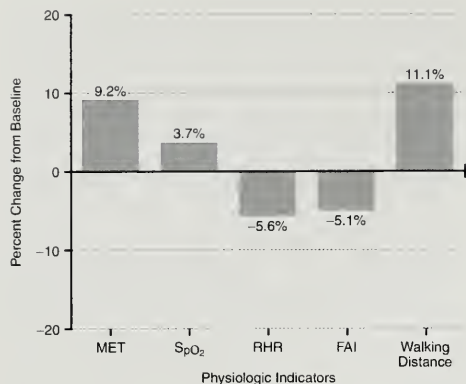


Fig. 2. The percent of change in physiologic indicators measured at the beginning and upon completion of the 7-week core program. The horizontal black line represents the baseline established at the beginning of the program. The bars represent the percentage of change from baseline. The indicators were MET = metabolic equivalents, SpO₂ = pulse oximetry oxygen saturation during exercise, RHR = resting heart rate, FAI = functional aerobic impairment, and walking distance all derived from a 6-minute walk test.

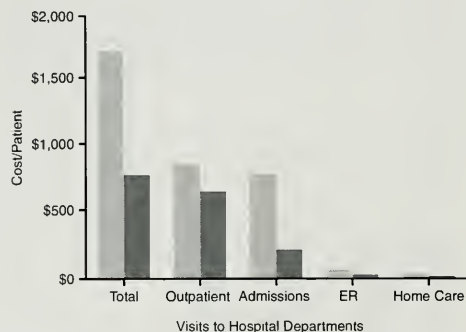


Fig. 3. Preprogram and postprogram charges per patient per year for the 47 patients in the study displayed in total and subtotals for outpatient visits, hospital admissions, emergency room visits, and home care visits. ■ = preprogram; ■ = postprogram. ER = emergency room.

bilitation Workshop where participants suggested the use of disease specific QOL indicators to possibly demonstrate improvement in patients' participating in a comprehensive program even before physiologic improvement is detected.¹³ The only QOL indicator in this study that did not improve significantly was the patients' ability to cope with the disease, perhaps a result of this program's lack of funding for social workers or spiritual counselors.

With the increasing regulatory pressure for health care organizations to assess QOL improvements,¹⁴ we believe de-

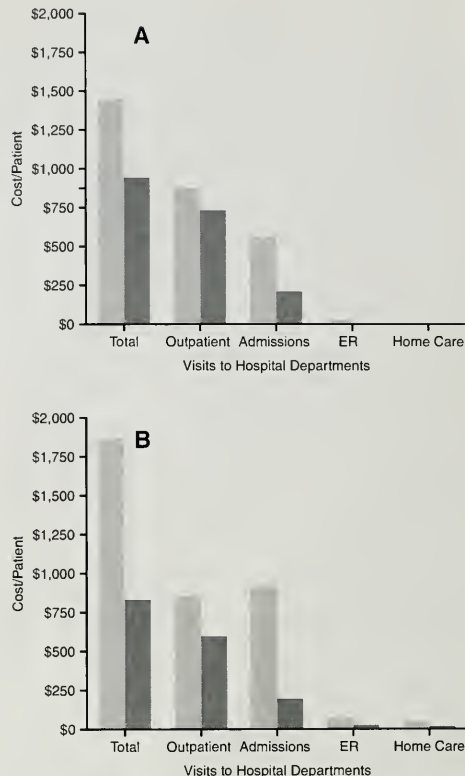


Fig. 4A. Preprogram and postprogram charges per patient per year for the Lung Gym group (n = 18) in the study displayed in total and subtotals for outpatient visits, hospital admissions, emergency room visits, and home care visits. B. Preprogram and postprogram charges per patient per year for the Non-Lung Gym group (n = 29) in the study displayed in total and subtotals for outpatient visits, hospital admissions, emergency room visits, and home care visits. ■ = preprogram; ■ = postprogram. ER = emergency room.

bate will continue over the most accurate way to measure such a subjective indicator. The short survey used in this study was developed internally to address the disease specific concerns of this particular group of patients. One study³ suggests that COPD patients be assessed for QOL indicators in addition to traditional clinical testing. The program should address the patient's 'fear of dyspnea.' This fear may worsen a patient's condition because the patient may avoid exertion, causing muscles to continue to weaken. There are several instruments that have been used,⁴ but the debate continues over whether to use the usual general health status questionnaires or whether disease specific surveys would be more meaningful.¹⁰ The Sickness Impact Profile was found in one study

to correlate better with the 6-minute walking distance than any spirometric measurements.¹⁵

The traditional focus of the health care industry on objective clinical indicators has resulted in a vast array of standard accepted methodologies for assessing improvements in health. While these measures continue to be useful, it is clear that they will have to be combined with QOL and utilization measures as the health care industry continues to evolve in response to patient pressure for satisfaction, payer pressure for reduced costs, and regulatory pressure for proof of quality.

Although it seemed that the Non-Lung Gym group had a larger decrease in costs, the fact that both the Lung Gym and the Non-Lung Gym groups demonstrated significant improvement supports the efficacy of the 7-week core component of the program at least through the first year after completion. This may have been due to the Lung Gym group's being more compliant, less ill, and/or better able to afford the Lung Gym fees. Such behavioral, severity of illness, and socioeconomic factors were beyond the scope of this study. Furthermore, ongoing exercise training has been recommended to avoid loss of improved muscle function and work performance and prolong the benefits derived from rehabilitation of COPD patients.¹⁶ Longer term differences between the groups remain to be studied.

Measuring utilization and calculating savings is perhaps the most challenging aspect of the new frontier in health care. Because health care is a service industry, it is difficult to identify all the actual costs associated with providing certain treatments. Complicating the task is the fact that the health care industry has not traditionally been concerned with controlling costs or keeping track of them. Billing systems have been designed to accommodate a FFS system based on exact coding and specific reimbursements for each service or diagnosis. Charge data were used in this study because actual costs were unavailable. A more thorough analysis would have been based on actual costs of the program including estimates of utilization savings in other areas where COPD was the secondary diagnosis. Other important considerations are the revenue that could be generated by offering the program to FFS patients, the value of the patients' improved QOL, the effect of improved care on customer retention, membership stability, and the pricing structure of HMO products, and, ultimately, the financial 'health' of the health care organization considering an investment in a program for pulmonary rehabilitation.

This program was designed and administered by a master's-level exercise physiologist and 2 registered respiratory therapists using 1,100 sq ft of space for 5 ergometers, 5 treadmills, and 2 upper body ergometers. The equipment cost approximately \$22,000 plus the cost of a set of free weights (1, 2, 3, and 5 lb), a scale, liquid oxygen, stopwatches, and a television. A quarter mile walking track was already available for walking tests and Lung Gym exercises. For 1996, operating expenses for the program were around \$74,000 resulting in a short-term profit margin of -30% but a long-term profit

margin of 30% which includes the 1-year postprogram utilization savings but excludes any reimbursements from the HMO. FFS patients contributed an average revenue of \$2,326/patient, while HMO patients contributed an average revenue of \$161 each in co-payments and Lung Gym payments.

Previous studies of utilization have focused on the duration and cost of hospital admissions for COPD patients^{3,4} resulting in estimates of savings at \$1,243 to \$3,817/patient per year.¹⁰ As the health care industry continues to evolve away from the FFS model of hospital-based care towards the HMO model of outpatient-based care with an emphasis on prevention and cost control, more research is needed in the area of outpatient utilization, perhaps including pharmacy costs which were not addressed in this study.

Conclusions

Overall, this study shows that improved QOL and physiologic functioning can result in decreased outpatient utilization for this patient population. This pulmonary rehabilitation program has been shown to be significantly beneficial to both the patients, subjectively and objectively, and to the health care organization. Even without the continuation of their rehabilitation that the Lung Gym offers, patients do benefit significantly from the program, at least within the first 12 months after completion of the core component. The benefits of such a program do not appear to be of only a short-term nature. Further study of this patient group or others like it is needed to demonstrate longer term benefits and savings.

PRODUCT SOURCES

NCSS 6.0, Number Cruncher Statistical Systems, Dr Jerry L Hintze, Kaysville UT

Ohmeda Biox Model 3740 Pulse Oximeter, Ohmeda, Louisville CO

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— Foreword —

The National Lung Health Education Program: A New Frontier for Respiratory Care Professionals

The National Lung Health Education Program (NLHEP) is a new health care initiative for America. NLHEP aims to identify smokers and patients with respiratory symptoms who are just beginning to develop airflow obstruction and are on the pathway to symptomatic chronic obstructive pulmonary disease (COPD).

The scientific foundations for NLHEP are found in the Lung Health Study.¹ Briefly, the Lung Health Study (LHS) identified nearly 6,000 people with mild degrees of airflow obstruction (mean age 48.5 ± 6.8 years, forced expiratory volume in the first second [FEV₁] of forced vital capacity [FVC] $63\% \pm 5.5\%$). The purpose of the LHS was to study strategies that could reduce the rate of decline in airflow obstruction as judged by FEV₁. In brief, those patients who succeeded in stopping smoking (22%) throughout the 5-year follow-up had an initial small improvement in FEV₁ followed by a very slight decline, resulting in essentially no losses in FEV₁ at the end of the 5-year follow-up. By contrast, the patients who continued to smoke within the 5-year period (78%) had more rapid rates of decline but were still not in the symptomatic range of COPD. Patients who were randomized to receive ipratropium bromide showed a bronchodilator effect throughout the 5 years of the study, but there was no change in baseline FEV₁. Another study, however, drew quite different conclusions, showing an improvement in FEV₁.²

An important outcome of the LHS was the cause of death which included lung cancer ($n = 57$), heart attack and stroke ($n = 37$), and all other causes of death including other smoking-related cancers. It is interesting that no patient died of COPD during the LHS for the simple reason that they had not yet reached the symptomatic range of FEV₁ and the stage of disease characterized by premature mortality. Thus, the LHS clearly showed that spirometric abnormalities are a surrogate marker for death from other major diseases.³ This fact has actually been well known since the Framingham Study. In fact, spirometric abnormalities predict all-cause mortality.⁴

NLHEP is directed to all primary care professionals including respiratory therapists, physician assistants, nurse practitioners, and primary care physicians. The aim of NLHEP is to reach all smokers and patients with common respiratory symptoms of dyspnea, cough, sputum, and wheeze, who are

already on the pathway to developing COPD but do not know this. In this regard, the respiratory care professional will likely play a key role. The major challenge in NLHEP is to put spirometers in the hands of all primary health providers and to promote accurate use of these devices. Respiratory care practitioners are knowledgeable in spirometry and in teaching proper spirometer techniques. It is hoped that the respiratory care professional can promote the widespread use of spirometry in all physician and other health care provider offices.

One of the challenges of NLHEP was to convince industry to develop a new generation of spirometers that are accurate, user friendly, essentially maintenance free with easy calibration, and low in cost. This was quite a challenge. However, at this writing at least 3 spirometer companies have met the challenge to develop such simple devices that give direct read-out of FEV₁, FVC, and the ratio between the two. The capability of printing out volume-time and flow-volume curves is an option, as is the level of memory for the spirometer. The office spirometer must become as ubiquitous as the sphygmomanometer for NLHEP to succeed.

NLHEP is sponsored by the American Association for Respiratory Care, the American Association for Cardiovascular and Pulmonary Rehabilitation, the American College of Chest Physicians, the American Thoracic Society, the American College of Physicians, and the Society of General Internal Medicine. Government sponsors are the Lung Division of the National Heart, Lung, and Blood Institute, the National Cancer Institute, and the National Institute of Occupational Safety and Health. Thus, both powerful societal and government agencies are allies for a common purpose.

In some ways, NLHEP is patterned after the National Hypertension, National Cholesterol, and National Asthma Education Programs. Certainly these 3 initiatives have had a major impact on the health of this country and have been particularly notable for the reduction in heart attack and smoking due to the control of major risk factors in cardiovascular diseases. The impact of the National Asthma Education Program is just being evaluated.

NLHEP, however, has an opportunity that goes beyond these previous health care initiatives. Because spirometric abnormalities are predictive of the risk of death from all causes

and, most particularly, lung cancer, heart attack, stroke, COPD, and other smoking-related cancers, the spirometer becomes the key instrument in finding patients for whom smoking cessation is an absolute must. It is fortunate that new knowledge concerning smoking cessation, new pharmacologic agents using various forms of nicotine replacements, and other pharmacologic agents including buspirone⁵ and bupropion⁶ can form an effective armamentarium that theoretically, at least, could result in 50% of smokers succeeding in sustained smoking cessation. Thus, NLHEP could be a major health care initiative that will go far beyond the challenge of COPD.

COPD, however, is the most rapidly rising cause of death—now ranking number 4 with an estimated 110,000 predicted deaths in 1998 alone. COPD alone is the only disease among the top 10 that continues to rise. Thus, COPD is a challenge for all health professionals, and the NLHEP offers a great new frontier for the respiratory care professional

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Strategies in Preserving Lung Health and Preventing COPD and Associated Diseases

The National Lung Health Education Program

The National Lung Health Education Program Executive Committee*

1. Background

In this issue of *RESPIRATORY CARE*, the following document is the resource for a new national health care initiative that is aimed at preventing or forestalling premature morbidity and mortality from chronic obstructive pulmonary disease (COPD) and related disorders. COPD is now the fourth most common cause of death in the United States and accounted for more than 105,000 deaths in 1997. COPD is the only major disease that is rising in prevalence and mortality; all other major causes of death are declining. Abnormal spirometry is a marker for not only COPD, but also for risk of lung cancer, heart attack, and stroke, as was learned from the Lung Health Study.¹

This is a major new education program, known as the National Lung Health Education Program (NLHEP).[†] NLHEP is aimed at a broad audience of clinicians who are in daily contact with smokers and people with chronic pulmonary symptoms. These clinicians include respiratory therapists, primary care physicians, and other front-line health care providers such as physician assistants, nurse practitioners, and others. This document is intended primarily for this audience. It offers in-depth advice on the early identification and intervention in COPD and related disorders. It also offers specific suggestions for the management of symptomatic patients with COPD. It is estimated that today (1998), approximately 200,000 primary care health providers see the great majority of smokers at risk of COPD and related disorders in their offices and clinics each day. These professionals are more

likely to be contacted by patients who smoke than are specialists. Every office visit is an opportunity to offer and encourage smoking cessation.

It has been estimated that 70% of smokers see a primary care clinician at least once each year for some reason.² The regular review of the smoking and brief symptom history, as well as use of the simple office spirometer, will identify patients who could be saved from late symptomatic stages of COPD by smoking cessation and other interventions. The key to diagnosis of all stages of COPD is simple spirometric measurements which can and should be used regularly in all patients at risk.

[†]The National Lung Health Education Program (NLHEP) is a new national health-care initiative aimed at early diagnosis and intervention in COPD and related disorders. The NLHEP is designed to reach all primary care practitioners and the patients they serve in a massive public health campaign for all smokers, former smokers, and any patient with pulmonary symptoms. The development of simple, practical, handheld spirometers will provide a new instrument to complement established pulmonary function testing so that all patients can "Test Your Lungs/Know Your Numbers," which is the battle cry of NLHEP.

The American College of Chest Physicians (ACCP) has formed an alliance with NLHEP and in turn will coordinate the supporting efforts of all other sponsors including the American College of Physicians, American Thoracic Society, Society of General Internal Medicine, American Association for Respiratory Care, and American Association for Cardiovascular and Pulmonary Rehabilitation. Government sponsorship is from the Lung Division of the National Heart, Lung, and Blood Institute, the National Cancer Institute, and the National Institute of Occupational Safety and Health. Together, medical professional societies and governmental agencies will join forces to curb the rise of COPD, now the fourth most common cause of death and the only major cause of death that is rising today. The NLHEP/ACCP alliance is timely and follows on the heels of the President's new initiative designed to control tobacco abuse among teenagers. This "strategic alliance" is well-positioned for success in preventing smoking, stopping smoking, and identifying patients on the pathway to premature morbidity and mortality from COPD and related disorders. The NLHEP resource document, authored by Executive Committee members of NLHEP, and sponsored by The Chest Foundation, was published in the February 1998 supplement to *CHEST*. For additional information, contact David Eubanks, EdD, Vice President, Education and Special Projects, or Mary Katherine Krause, Associate Vice President, Education at (847) 498-1400 or e-mail deubanks@chestnet.org.

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2. Introduction

The drama that surrounds sudden and life-threatening illness such as heart attack and stroke has resulted in the combined efforts of the public, primary care, and specialist physicians to devise effective strategies for prevention or early treatment of these common disabling and often fatal diseases. By contrast, the same "call to action" is not present in COPD. Accordingly, early and aggressive preventive management has not, thus far, been given the same urgent priority. This may be due largely to the lack of compelling symptoms or no symptoms at all during years of progressive loss of lung function on the pathway to disabling dyspnea and premature mortality from acute and chronic respiratory failure.

The major reduction in premature morbidity and mortality from heart attack and stroke can be attributed to an increase in physician and public awareness of the role of undetected and undertreated hypertension and high blood cholesterol levels as major risk factors in these lethal or disabling cardiovascular disease states. Recent evidence has demonstrated that during the two decades 1971 to 1991, patients' awareness of hypertension has increased markedly from 51 to 84%.³ Concomitantly, the percentage of patients treated for hypertension increased from 36 to 83%, although the achievement of ideal BP (blood pressure) has not been very satisfactory, ie, only 21%.³ Still, most patients are aware of "their numbers" and can usually recite their usual levels of both systolic and diastolic BP to health care workers, family, and friends. Most can name the antihypertensive medication(s) that they take and many are aware that smoking, excess alcohol, and overweight along with excessive sodium and fat consumption are risk factors for hypertension and related heart attack and stroke. Most patients are also aware of the "cholesterol numbers" and recognize the difference between "bad" (low density) lipoproteins and "good" (high density) lipoproteins. A major reduction in cholesterol levels during the past 30 years through diet, weight loss, and medications has also played an important role in reducing the prevalence of heart attack and stroke.⁴

Certainly a reduction in smoking prevalence, which began with the first Surgeon General's Report on the health hazards of smoking in 1964, has played an important role in reducing the morbidity and mortality of heart attack and stroke. The control of these major cardiovascular risk factors today forms the basis of early identification of life-threatening abnormalities at a time when preventive measures can be instituted. Improvements in the management of symptomatic ischemic and thrombotic cardiovascular disease have also contributed to a reduction in heart attack and stroke. These advances can be attributed in large part to advances in understanding the causes, pathogenesis, diagnosis, and treatment of cardiovascular diseases and to national health care education programs sponsored by the National Heart, Lung, and Blood Institute.

In the field of pulmonary medicine, a major National Asthma Education Prevention Program has also aroused both physician and patient awareness of the nature of asthma and how early treatment can prevent the loss of quality of life or even prevent fatal attacks.⁵ This progress has resulted from a shift of emphasis away from the use of bronchodilating agents alone to new strategies in preventive management aimed at suppressing the inflammatory component of disease. Thus, anti-inflammatory agents administered by the inhaled route have achieved the status of first-line or cornerstone drugs in asthma maintenance management.^{5,7} There is also a growing consensus on strategies for the management of severe exacerbations of asthma.⁸ A global initiative for asthma identification and management has been developed in conjunction with European physicians and those from elsewhere in the world.⁹ Utilization of home peak flow measurements has added a new dimension to the monitoring of variations in airflow as an index of asthma stability and severity and responses to bronchoactive drugs.

Unlike the ubiquitous use of the sphygmomanometer in the offices and clinics of virtually all health care providers and in drug stores and grocery stores, today there is only limited use of simple spirometric measurements for the detection of asymptomatic stages of COPD or to gain objective measures of severity in symptomatic patients, as well as to document responses to therapy. Very few patients with COPD know the importance of testing their lungs or can recite "their numbers" for FEV₁ (forced expiratory volume in the first second) and FVC (forced vital capacity).

With publication of the results of the Lung Health Study, a new scientific basis for early identification of and intervention in COPD has now been established.¹ Several key points should be specified. Identification of mild but clinically significant airflow abnormalities in a young to middle-aged population is possible by spirometry. The excessive rate of decline in FEV₁ over 5 years can be virtually stopped by smoking cessation, compared with accelerated losses in FEV₁ in continuing smokers. Of paramount importance is the fact that even mild airflow abnormalities signal a high risk of death from lung cancer, heart attack, and stroke. Thus, it appears that the progress of these major diseases often can also be reduced by smoking cessation. In the Lung Health Study, only 22% of patients randomized to special care succeeded in stopping smoking for the full 5 years of the study; only 5% were successful if randomly assigned to usual care.¹

Thus, prevention and management of COPD must go beyond smoking cessation, although freeing patients from tobacco use should always be the first priority. Today a large armamentarium of therapeutic and preventive agents is available to deal with both asymptomatic and symptomatic patients with COPD. These therapies, along with smoking cessation, must first be offered and supervised by primary care physicians and other "front-line" health care providers working in part-

nership with the educated patient. How these treatment options can be applied is covered in the pages that follow.

As this new initiative, the NLHEP, develops and evolves, it is likely that a worldwide consensus on COPD identification and management will follow.⁹ This document ushers in a new effort and era in the identification and treatment of early stages of COPD and associated diseases.

3. Definitions

COPD is an all-inclusive and nonspecific term that refers to a defined set of breathing-related symptoms:^{10,11} chronic cough, expectoration, varying degrees of exertional dyspnea, and a significant and progressive reduction in expiratory airflow. Most patients with COPD are smokers. This airflow abnormality does *not* show major reversibility in response to pharmacologic agents. Hyperinflation and a reduced diffusing capacity may be present. Inflammatory damage to both airways (asthmatic and chronic bronchitis) and alveoli (emphysema) are found at postmortem examination.

COPD is an umbrella term used to encompass several more specific respiratory diagnoses that may occur individually or in any combination. The other terms—chronic obstructive airways disease, chronic obstructive lung disease, chronic airflow (or airways) obstruction, and chronic airflow limitation—all refer to the same disorder. The specific components within the COPD designation are chronic bronchitis, emphysema, and asthmatic bronchitis. Overlaps are common.

4. Pathophysiology/Risk Factors

Pathophysiology

Limitation of airflow can result from several pathophysiological mechanisms.¹²⁻¹⁵ Contraction of airway smooth muscle can narrow airways and obstruct airflow. This mechanism likely plays an important role in asthma and may also contribute to airflow obstruction in other forms of COPD. Narrowing of airways can also be affected by inflammation, edema, and peribronchiolar fibrosis that can both distort and narrow the airways.¹⁵ Mechanisms that cause these inflammatory changes may be different from the cytokine-mediated inflammatory changes in asthma airways. It is a fact that smoking causes these changes, probably via elastase and oxidative mechanisms, but the details are not precisely known. Destruction of alveolar walls and loss of elastic recoil are the defining features of emphysema.^{15,16,16a} As a result, both driving pressure and intraluminal pressure maintaining the patency of small airways are reduced. This may play a particularly important role in patients with severe disease.^{16,16a} Finally, the airways may be occluded by secretions. While this mechanism may not contribute to fixed airflow obstruction, secretions generally play a prominent role in acute exacerbations of both asthma and chronic bronchitis.

Risk Factors

Certain risk factors besides smoking have been identified for COPD. Genetic background predisposes some individuals to the development of asthma. Genetic deficiency of α -1-protease inhibitor contributes importantly to the development of emphysema in a small percentage of patients.¹⁷ Family clustering of COPD also exists in the absence of the α -1-antiprotease deficiency state.¹⁸

Cigarette smoke plays a prominent role and is undoubtedly the major risk factor for the development of emphysema and chronic bronchitis. It is likely that tobacco smoke can contribute to the development of COPD through several related mechanisms. For example, many of the more than 4,000 components of cigarette smoke are active oxidants. These species can oxidize the methionyl residue at the active site of an α -1-protease inhibitor, resulting in an acquired form of protease inhibitor function.¹⁹ Components of smoke can also activate complement and inflammatory cells²⁰ and may be able to inhibit lung repair mechanisms.²¹

Other exposures may also play a role. Occupational exposures to dusts and some fumes are important factors contributing to chronic bronchitis. The clinical importance of coal dust exposure has been carefully studied in British miners.²² There was no evidence of synergism between smoking and dust exposure.

Other contributing factors in COPD include air pollution, childhood respiratory tract infections, and nonspecific bronchial hyperreactivity. These factors have been discussed in great detail in a most comprehensive chapter by Buist and Vollmer.²³

Grain dust exposure has also been established as a risk factor in COPD, both in smokers and nonsmokers. Some of the best studies came from Canada.²⁴ The mechanisms appear to be both immunologic and nonimmunologic; bronchial hyperresponsiveness is quite prevalent after grain dust exposure in susceptible individuals. Endotoxins, mites, and fungi may also be responsible for the effect on airflow obstruction and bronchospasm.²⁴

Air pollution has been causally linked with COPD, but its role has been considered minor as compared with that of smoking. Bates²⁵ produced some of the most convincing evidence in his study of veterans with equal smoking histories in four Canadian cities. Three cities had significant air pollution and one (Winnipeg) was relatively free of air pollution. Chronic bronchitis was more prevalent in the polluted than the non-polluted cities.²⁵ Another report related photochemical oxidants and multiple primary air pollutants such as sulfur dioxide particles and hydrocarbons to chronic respiratory symptoms and pulmonary function abnormalities in both smokers and nonsmokers.²⁶ Many more references to the association between air pollution and COPD have been made.²³

Respiratory symptoms have been related to the use of several domestic cooking fuels, such as kerosene and other fuels in India,²⁷ and wood burning for cooking and heating in Bogota,

Columbia.²⁸ Indoor tobacco smoke pollution has been shown to have adverse effects on pulmonary function in children and adolescents.²⁹ A deleterious effect of low socioeconomic status was found in studies that controlled for smoking intensity, but the effect on pulmonary function was weak as compared with smoking.³⁰

A unique and comprehensive study on the epidemiology, course, and prognosis of COPD gave evidence of childhood respiratory infections as a risk factor in COPD.³¹ The presence of repeated childhood respiratory infections was associated with relatively mild impairment in ventilatory impairment in young adults. Such patients demonstrated an accelerated rate of decline in lung function with advancing years and cigarette use. It is possible to conclude from this study that damage to young developing airways sets the stage for more aggressive injury from tobacco smoke in later years.^{31,32}

Reports on the association between alcohol consumption and the prevalence of COPD vary. Heavy alcohol consumption was associated with respiratory symptoms and reduced lung function in the study by Lebowitz,³³ even when controlled for smoking. But smoking was judged to be a far more important risk factor. The effect of passive smoking would also have to be considered in any heavy drinker, smoker or not, who consumed his or her favorite beverage in heavily tobacco-polluted bars or restaurants. By contrast, no association between alcohol consumption and COPD was found in the study by Higgins et al.³⁴

A carefully designed and administered questionnaire, along with biochemical tests, has shown a much lower food intake and serum levels of the major antioxidant vitamins in smokers as compared with nonsmokers.³⁵ Because oxidant injury is believed to be one of the mechanisms of alveolar and airway damage, perhaps a deficit in antioxidants in food could be a risk factor. If so, this could be an important factor in the development of COPD in areas of the world where malnutrition is prevalent. New evidence relates a low intake of vitamins C and E (alpha tocopherol) to a reduction in FEV₁ in elderly people.³⁶

While there are several clearly defined risk factors that predispose to the development of COPD, not all individuals with similar exposures develop disease. It is unclear, at present, why only 10 to 20% of cigarette smokers (of one pack per day or more), for example, will eventually develop clinically significant airflow limitation. Airflow limitation generally progresses slowly over an extended period of time.³⁷ Individuals who have developed airflow limitation, however, are clearly at risk to continue to lose function and to develop clinically significant disease. This prolonged natural history presents the opportunity of asymptomatic case identification and intervention. It is rational, therefore, to attempt to identify such individuals by the use of screening spirometric measures. Simple tests such as measurement of FVC and FEV₁ can easily be performed with simple equipment in any physician's office. Results of such tests may be abnormal for a number of rea-

sons, but reduced function is clearly associated with an increase in mortality from all causes.³⁸ Serial determinations of spirometry findings, therefore, should be regarded as part of routine general screening that offers patients a number of potential benefits. In this regard, detection of reduced expiratory airflow can be used as an educational tool to improve smoking cessation rates.³⁹

5. Magnitude of the Problem and Socioeconomic Differences

In the United States, COPD is the fourth leading cause of death after heart disease, cancer, and stroke.⁴⁰ These diseases are present in nearly 16 million persons, and in 1993, there were > 500,000 hospitalizations, 14 million physician office visits, and nearly 96,000 deaths from COPD, excluding asthma (Tables 1 and 2). Prevalence and mortality have been increasing appreciably and in contrast to many leading chronic diseases.⁴¹ As life expectancy increases, greater prevalence and mortality from COPD are likely. The main diagnostic components of COPD are emphysema and chronic obstructive bronchitis. Emphysema, the more serious, is a particularly disabling and progressive disease. Chronic obstructive bronchitis is highly prevalent and a major cause of restricted activity, chronic disability, and lost productivity from youth to old age. In 1993, morbidity and mortality from COPD cost the nation \$14.7 billion in medical care expenditures, \$6.1 billion for hospital care, \$4.4 billion for professional services, \$2.5 billion for drugs, \$1.5 billion for nursing home care, and \$1.0 billion for home care.⁴¹⁻⁴⁵

"Chronic obstructive pulmonary disease" is a nonspecific term for "diseases of uncertain etiology characterized by persistent slowing of airflow during forced expiration."⁴⁶ In the terminology of the *International Classification of Diseases (ICD), Ninth Revision*, it includes: Bronchitis, not specified as acute or chronic, Chronic Bronchitis, Emphysema, and Chronic Airways Obstruction, not elsewhere classified (Table 1).

For most national statistics, Bronchiectasis (*ICD* code 494) and Extrinsic Allergic Alveolitis (*ICD* code 495) are included, but these relatively uncommon conditions have virtually no effect on the size and trend of the morbidity and mortality statistics for COPD. Asthma (*ICD* code 493) is not included with COPD, except when the category "Chronic obstructive pulmonary disease and allied conditions" (*ICD* codes 490-496) is used for international mortality comparisons, since asthma mortality is not reported separately.

Morbidity

From self-reported conditions reported in the 1993 National Health Interview Survey, it is estimated that 13.8 million Americans have chronic bronchitis and 2 million have emphysema.⁴³ Although some people report both conditions, these estimates of prevalence are underestimates since as many as 50% of cases

Table 1. Morbidity and Mortality from Chronic Obstructive Pulmonary Disease (COPD) in the United States, 1993*

	ICD9 Codes	Deaths	Hospitalizations (thousands)	Doctor's Office Visits (thousands)	Prevalence (millions)	%
Chronic bronchitis	490,491	3,500	265	10,577	14	5.4
Emphysema	492	17,990	37	494	2	0.8
Other chronic airways obstruction	494-496	74,320	203	3,187	—	—
Total COPD	490-496	95,900	505	14,258	16 [†]	6.2 [†]

*Source: National Center for Health Statistics: Vital Statistics, National Hospital Discharge Survey, National Ambulatory Medical Care Survey, and the National Health Interview Survey.

†Includes double counting of persons with both conditions.

Table 2. Mortality Rates from Chronic Obstructive Pulmonary Disease (COPD) by Education and Income*

	Men		Women	
	45-64 years	65+ years	45-64 years	65+ years
Education (highest grade completed)				
< HS	49	345	27	101
Completed HS	24	237	11	102
> HS	15	232	8	91
Family Income (\$1980)				
\$0-10,000	85	378	27	111
\$10-24,999	28	256	18	98
\$25-49,999	13	229	7	83
≥ \$50,000	4	166	5	54

*Age- and race-adjusted annual death rates per 100,000. COPD includes ICD9 underlying cause of death codes as 490-492 and 496. HS = high school. Source: National Longitudinal Mortality Study.

are undiagnosed and are thus omitted. Thus, COPD may be present in > 30 million Americans. The prevalence of chronic bronchitis apparently increased from 3.3% of the population in 1970 to 5.4% in 1993.^{43,44} The prevalence, based on self-report, is similar across all age groups, about 4 to 5%, but it is higher in women than men and higher in the white than the black population.⁴¹ In contrast to COPD mortality trends shown later, there are no marked age or sex differences in time trends for prevalence of chronic bronchitis. The prevalence of emphysema is much higher in men than women and higher in whites than blacks, and the trend has been essentially flat over the 1970 to 1993 period.^{41,43,44}

The annual number of days of restricted activity reported during the 1986 to 1988 National Health Interview Survey was over 114 million for chronic bronchitis and emphysema, ranking these conditions among the highest 10 chronic conditions for this measure of morbidity in the nation.⁴⁵ Restricted activity days amounted to about five per year for persons with chronic bronchitis and 28 per year for persons with emphysema. There were no major differences by sex. The annual number of disability days was 27 million for chronic bronchitis, 2.2 days per condition per year, and 26 million for emphysema, 13.1 days per condition per year.

Mortality: Magnitude and Trend

The age-adjusted death rate for COPD is 60% higher in men than in women.⁴⁰ The death rate is higher in black men than white men until about age 60 years, after which the rate is much higher in whites.⁴¹ The age-adjusted death rate continues a long-term increase (Fig 1). Mortality from the cardiovascular diseases and COPD increased in the 1950s and into the 1960s. As mortality decreased markedly for the cardiovascular diseases beginning in the mid 1960s, COPD mortality continued to increase. The age-adjusted death rate doubled between the 1955 to 1957 period and the 1957 to 1963 period and also between the 1957 to 1963 period and the 1963 to 1983 period: it increased 22% from 1983 to 1993.⁴¹ In the 1960s, the increase was much greater in men than women.⁴¹ By the 1980s, however, the increase was strikingly greater in women than men and greater at older rather than younger ages. Since 1980, the age-adjusted death rate for COPD has leveled off in white men, increased in black men, and increased markedly in white and black women.⁴¹ A peak in the trend was reached in the 1980s in men for each age group except 85 years and older, and a peak was reached in women, but only in those younger than 55 years of age (Fig 2).⁴¹ With

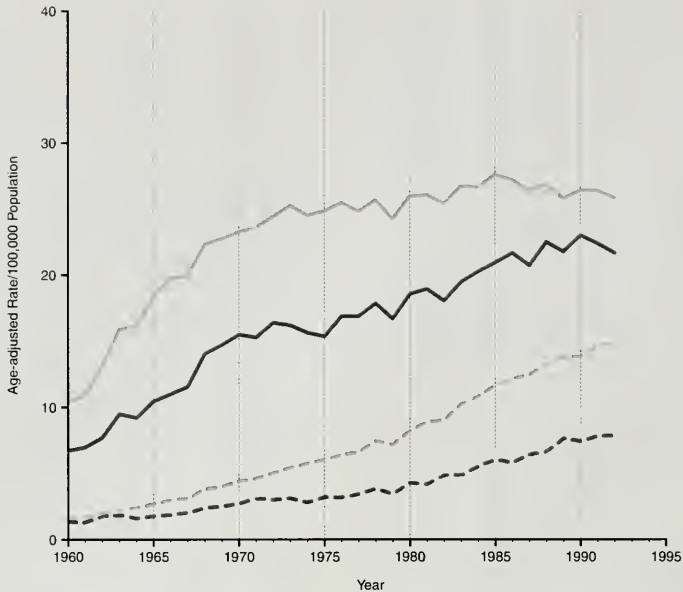


Fig. 1. Death rates for chronic obstructive pulmonary disease by sex and race, United States, 1960-1992. — = White men; --- = White women; — = Black men; --- = Black women. Nonwhite from 1960-1967. ICD codes 490-492, 494-496 (excludes asthma). Source: Vital Statistics of the U.S.

respect to geographic differences, the Western mountain states have the highest COPD mortality, followed by the Eastern mountain states.^{41,46} Among 32 industrialized countries, the United States ranks 14th highest in mortality from COPD and allied conditions for men and eighth highest for women.⁴¹⁻⁴⁷

Impaired Pulmonary Function

When completed, data from the Third National Health and Nutrition Examination Survey will provide current estimates of the prevalence of impaired pulmonary function based on measurements in a nationally representative sample of black, white, and Mexican-American men and women for whom extensive data on pulmonary, cardiovascular and other diseases, smoking habits, and other characteristics will also be available.

COPD by Socioeconomic Status

Mortality rates from COPD are strongly related to socioeconomic status (Table 2). In the National Longitudinal Mortality Study, education, income, and occupation are determined from the Current Population Survey and the individuals in the survey are followed for mortality using the National Death Index. Very strong relationships between low family income and death from COPD are observed for both men and women

in the age group 45 to 64 years.⁴⁶ Relationships are weaker, though still very strong, for the age group 65 years or older. Mortality rates from COPD are threefold higher for those in the age group 45 to 64 years with less than a high school education as compared to those with more than a high school education. Since there are fewer women and older persons with occupations, occupation data are available only for men in the age group 45 to 64 years. There is a clear increase in mortality from COPD in those with blue collar occupations. For national morbidity data, the 1993 National Health Interview Survey reported an inverse relationship between prevalence of chronic bronchitis and emphysema and family income for ages 45 to 64 years and 65 years and older.⁴³

Cigarette Smoking

Trends in cigarette smoking are shown in Figure 3, where age-adjusted prevalence rates are plotted for white and black men and women, from 1965 to 1994. The decline in cigarette smoking has been greater in men than women, and greater in those with more years of formal education. The demographic patterns and trends for this major cause of COPD are reflected in the patterns and trends for COPD mortality and morbidity and identify opportunities for prevention and treatment of these diseases.

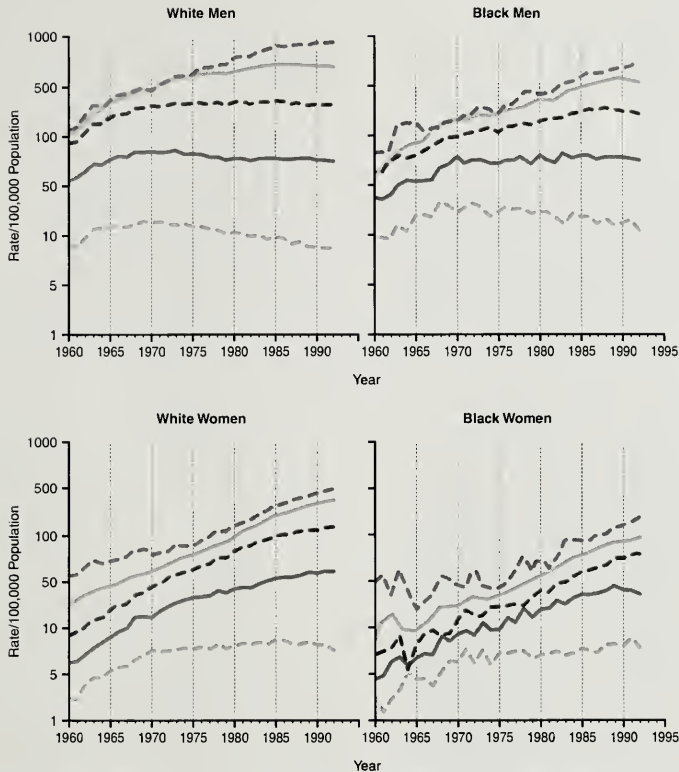


Fig. 2. Death rates for chronic obstructive pulmonary disease by age, United States, 1960-1992. — = 45-54 years; — = 55-64 years; — = 65-74 years; — = 75-84 years; — = ≥ 85 years. Non-white from 1960-1967. Source: Vital Statistics of the U.S.

6. Natural History

Early Airflow Decline as a Predictor of COPD

The natural history of COPD is characterized by slowly progressive limitation of expiratory airflow. This can be well assessed by simple spirometry and only two parameters, the FEV_1 and the FVC, need to be measured to obtain almost all the useful information available.⁴⁸ Variations in natural history are probably related to differences in the dose and influence of various risk factors. Beyond question and by common consensus, the most important risk factor is *cigarette smoking*. Fortunately, it is also the one that has been most completely studied.^{23,49} We know that only a certain percentage of cigarette smokers develop COPD or, stated another way, have progressive decline of airflow. This percentage is estimated to be somewhere between 10% and 30%.³⁷ There

are a number of hypotheses as to why only certain smokers are susceptible, but currently there is no uniform consensus. The Dutch hypothesis is perhaps the most long-standing, and implies that the smokers who are at risk are either atopic or have clinical or subclinical asthma, which is modified by exposure to cigarette smoke.⁵⁰ While this is an intriguing possibility, it is by no means completely accepted. There is agreement by most that bronchial hyperactivity ("twitchy" airways often demonstrated by serial spirometry taken after increasing doses of inhaled material, such as methacholine, that is likely to trigger bronchospasm in susceptible individuals) is a risk factor for COPD.⁵¹ But, it is not known whether this occurs because cigarette smoking causes bronchial hyperactivity in some individuals who then are at greater risk because of the combined risk factors, or whether the bronchial hyperactivity preexists prior to a patient becoming a cigarette smoker.

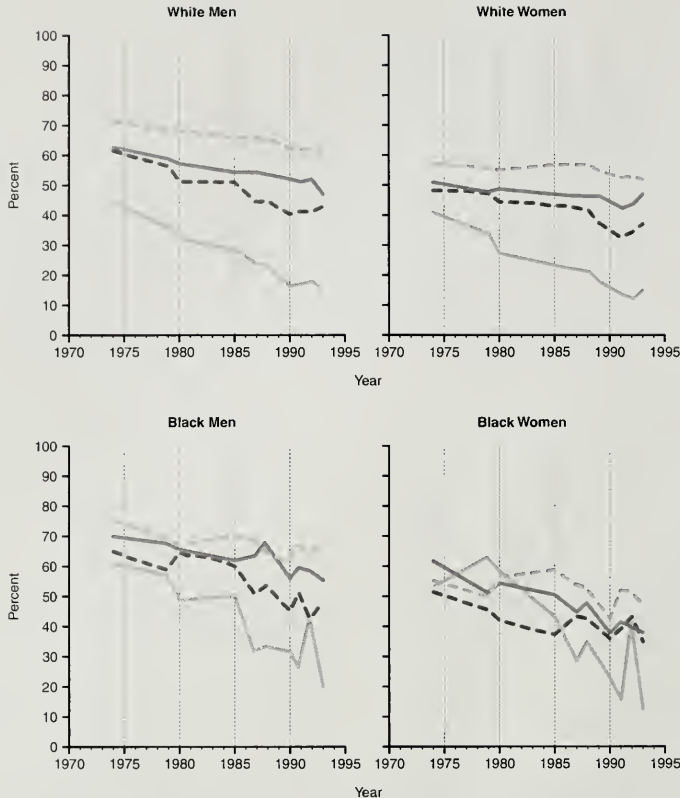


Fig. 3. Age-adjusted prevalence of cigarette smoking: ages 25 years and older, by education; United States, 1974-1993. — = < 12 years; — = 12 years; ■ = 12-15 years; — = ≥ 16 years. Source: National Health Interview Survey.

It is quite clear that an excellent predictor of continued airflow decline is evidence of decline early in a cigarette smoker's exposure. Fletcher et al³⁷ pointed out that one could predict with reasonable accuracy those patients who would develop severe COPD by identifying younger smokers who are just beginning to have early airflow obstruction.

Early Screening and Intervention

Because there is such a large reserve of pulmonary function, this deterioration in airflow obstruction can proceed undetected for years if pulmonary function tests are not done. In fact, except in those individuals who engage in vigorous exercise, quite severe airflow obstruction is often present before any symptoms of COPD develop. While we have known for some time that the natural history of COPD is that of pro-

gressive airflow deterioration, we had not known for sure that intervention through smoking cessation could modify the natural history of this disease. We have known, of course, that cigarette smoking is quite important in the development of many other diseases. Smoking cessation in individual patients has been documented to have positive health effects in almost all of these diseases.⁵² The Lung Health Study was the first prospective demonstration in a large population of relatively young smokers with only mild to moderate degrees of airflow obstruction that smoking cessation could modify the rate of decline of FEV₁.¹

Interventions with bronchodilator therapy and with anti-inflammatory therapy have been proposed⁵³ as possibly interfering with the natural history of this disorder, but there is no real documentation of this. In fact, the Lung Health Study II^{1,54} and the Euroscope Study are currently evaluating the effect

of baseline inhaled corticosteroids on the course and prognosis of COPD.⁵⁵ The Lung Health Study suggested that bronchodilator therapy, in the form of ipratropium bromide, does not modify the course of airflow decline even though there is a potentially useful and prolonged pharmacologic effect that is undiminished over 5 years.¹ This effect appears to be primarily that of bronchial airway dilation while the drug is present, as opposed to modification of the rate of deterioration in airflow in baseline lung function. By contrast, another study of ipratropium did show a favorable effect on baseline spirometry results over time compared with β -agonist therapy.⁵⁶

Oxygen therapy in stable hypoxic individuals with COPD has been shown to increase the life span of these patients when oxygen deficiency is corrected (see Section 10, "Strategic Drugs"). The improvement, however, only parallels the life expectancy that one would expect in individuals with comparable levels of airflow obstruction who did not have hypoxemia and, therefore, did not need oxygen therapy. So, in a sense, any impact on the natural history of the disease by oxygen therapy relates only to hypoxemia, not to progressive airflow deterioration, which most agree to be a better marker of the natural history of the disease. In any case, oxygen therapy is a late intervention for advanced COPD and it remains expensive.

Variations in the Smoking Population

The progressive increase in airflow obstruction characteristic of COPD has historically been thought of as affecting susceptible individuals in the smoking population, as if the smoking population could be clearly separated into susceptible smokers and all other smokers. An apparent dichotomy can be created by taking those who have the most severe disease and comparing their mean decline in pulmonary function with all other smokers. When one does this, it appears that those at risk of COPD have a decline in their FEV₁ at the rate of about 70 mL/year, and the others decline at about the rate of 20 mL/year.³⁷ There are many individuals whose FEV₁ decline at intermediate rates between these two means and even, of course, outside the two population means. So every individual does not fall neatly into one of two completely distinct populations. Nevertheless, there are clearly two extremes—cigarette smokers who decline very little in FEV₁, and those who decline very rapidly; of course, there are some who fall in between. Since the variations in FEV₁ decline relate to variations in susceptibility that probably can be accounted for by risk factors other than smoking (some of which have been described and studied and some that have not yet been thought of), this will be a fruitful area of further investigation. Those smokers without a significant decline in pulmonary function are still at great risk of developing many of the other smoking-related disorders, and should not at all be reassured of the safety of continuing to smoke. The mechanisms of protection from COPD from

smoking in most smokers are not well established. One possibility may be an oxidant/antioxidant imbalance in smokers who develop COPD.^{36,57}

Early Identification

At the present time, however, we have enough information to identify the approximately 20% of cigarette smokers who are experiencing a rapid fall in spirometric function over time. These people already have early airflow obstruction and, within that group, represent almost all of the patients who will someday develop severe COPD. They can be detected at an early stage through simple spirometry long before symptoms develop.¹ To add further interest to this group, it is generally agreed that those smokers who develop severe airflow obstruction (when compared with other smokers) are at greater risk of developing a number of other very important diseases. These diseases include coronary artery disease,⁵⁸ lung cancer,⁵⁹ and stroke,⁶⁰ all of which can be prevented or modified by a number of measures, the most important being smoking cessation. While smoking cessation is important for all smokers, this is a group of patients in whom large expenditures on very aggressive smoking cessation efforts can easily be justified. Certainly, an understanding of the natural history of COPD allows us to identify at an early stage a population not only at great risk of dying of COPD, but also at risk of dying of the three more common major causes of death: heart disease, cancer, and stroke. Over time, a significant number of patients with early stages of COPD die of heart disease, lung cancer, and stroke before they get to the stage of symptomatic COPD. Thus, competing causes of mortality prevent the development of symptomatic COPD. Understanding the natural history of lengthy stages of COPD and intervening early could become the cornerstone of a very important new public health program, with an impact on the *four leading causes of death* in the United States. The key to success is early identification and intervention via smoking cessation, which can favorably impact on all of these common causes of death.

7. Differential Diagnosis

Signs and Symptoms

Significant overlaps exist in signs and symptoms among the three major diseases of airflow obstruction: asthmatic bronchitis, chronic bronchitis, and emphysema. The large overlap has been long noted and well illustrated in Venn diagram fashion (Fig 4).¹¹ Nevertheless, signs and symptoms of COPD have been well characterized and can be identified through appropriate history and physical examination. While acute, episodic, or even perennial but reversible asthma does not strictly fall under the category of COPD since its hallmark is reversible airflow obstruction, it will be considered briefly here to contrast it with COPD which, despite its chronic and

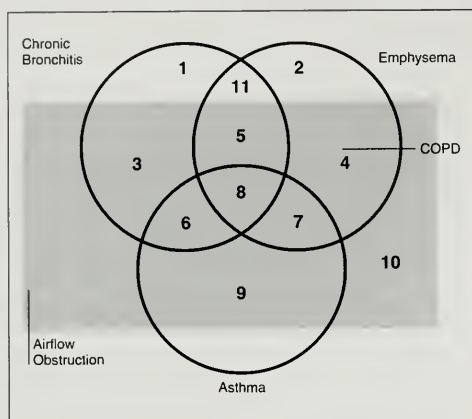


Fig. 4. Schema of chronic obstructive pulmonary disease (COPD). This nonproportional Venn diagram shows subsets of patients with chronic bronchitis, emphysema, and asthma. The subsets comprising COPD are shaded. Subset areas are not proportional to actual relative subset sizes. Asthma is by definition associated with reversible airflow obstruction, although in variant asthma, special maneuvers may be necessary to make the obstruction evident. Patients with asthma whose airflow obstruction is completely reversible (subset 9) are not considered to have COPD. Because in many cases it is virtually impossible to differentiate patients with asthma whose airflow obstruction does not remit completely from persons with chronic bronchitis and emphysema who have partially reversible airflow obstruction with airway hyperreactivity, patients with unremitting asthma are classified as having COPD (subsets 6, 7, and 8). Chronic bronchitis and emphysema with airflow obstruction usually occur together (subset 5), and some patients may have asthma associated with these two disorders (subset 8). Individuals with asthma exposed to chronic irritation, as from cigarette smoke, may develop chronic productive cough, a feature of chronic bronchitis (subset 6). Such patients are often referred to in the United States as having asthmatic bronchitis or the asthmatic form of COPD. Persons with chronic bronchitis and/or emphysema without airflow obstruction (subsets 1, 2, and 11) are not classified as having COPD. Patients with airway obstruction due to diseases with known etiology or specific pathologic condition, such as cystic fibrosis or obliterative bronchiolitis (subset 10), are not included in this definition.

progressive nature, is also characterized by exacerbations and remissions in the degree of airflow obstruction. In fact, many of the signs and symptoms of COPD are nonspecific and can also occur in other disease states such as congestive heart failure and vocal cord dysfunction.^{61,62}

Symptoms

Asthma. "Pure" asthma is characteristically episodic in nature with periods of normal airway function between acute obstructive exacerbations. While this episodic nature is the rule, some patients have prolonged exacerbations and may be left with

some degree of chronic airflow obstruction between acute episodic attacks. Asthma may begin in childhood or later in life. Adult-onset asthma often, but not always, occurs in people who had asthma in childhood and experienced an asthma-free period of years in their teens and early adulthood. In either case, exacerbations often have identifiable triggers that include cold air, exercise, inhaled irritants, allergens, respiratory infections, or emotional factors.

In the steady state, asthmatics are asymptomatic and can have normal expiratory flow rates.¹¹ Asthmatic attacks are characterized by dyspnea and wheezing. Cough is common and may, on occasion, be the sole symptom of asthma.⁶³ When the attack is severe, patients will often sit up leaning forward with their arms elevated and resting on a table to increase their inspiratory volume. In states of chronic symptomatic asthma (asthmatic bronchitis), exacerbations and remissions are also common, but airflow obstruction to some degree is always present. Patients with chronic symptomatic asthma, with even mild degrees of airflow obstruction while receiving therapy, are included in the umbrella designation of COPD.

Chronic Bronchitis. The hallmark of chronic (obstructive) bronchitis is considered to be morning cough productive of sputum for at least 3 months a year for a minimum of 2 years. These symptoms have been found to correlate directly with chronic inflammation of large airways⁶⁴ and inversely with FEV₁.⁶⁵ It is true, however, that people may have such symptoms without a reduced FEV₁.²⁵ This is called simple bronchitis and is not part of the COPD designation. Unlike in asthma, the symptoms are gradual in onset.

In the steady state, patients with chronic obstructive bronchitis have sputum production and, usually, some degree of exercise limitation or chronic dyspnea. Exercise-related symptoms may be so gradual in onset that patients may be unaware of them unless specifically questioned about detailed changes in activity over time. It is often helpful to ask about specific activities such as walking, stair climbing, shopping, or recreational exercise such as golf.

Exacerbations, often called "asthmatic bronchitis," are usually triggered by infections and are characterized by increased dyspnea with wheezing and increased cough. In advanced disease, patients may not experience dyspnea, presumably due to a reduced ventilatory drive, but they may have symptoms related to CO₂ retention (drowsiness) and right ventricular failure (edema). They may also have symptoms, such as headaches and daytime somnolence, related to sleep-disordered breathing.

Emphysema. Emphysema is generally a destructive rather than an inflammatory process and so is not usually associated with cough and sputum production. In the steady state, patients are usually dyspneic, often at rest and almost always with exercise. As with chronic bronchitis, the symptoms are gradual in onset.

Exacerbations are often associated with episodes of respiratory infection and are characterized by increased dyspnea. However, most patients with COPD have varying degrees of airway inflammation and remodeling (chronic bronchitis) and emphysema. Thus, the clinical manifestations include features of both, most commonly.

Signs

Asthma. Results of the physical examination should be normal in the steady state between attacks. During attacks, patients have a prolonged expiratory phase and wheezes.⁶⁶ During severe attacks, air movement may be too poor to cause wheezes. The chest may be hyperresonant to percussion and diaphragmatic excursions may be limited. Severe attacks are usually associated with cyanosis, tachypnea, tachycardia, intercostal muscle retractions, use of accessory muscles, and pulsus paradoxus. The need for hospitalization has been associated with heart rate > 120 beats/min, respiratory rate > 30 breaths/min, a pulsus paradoxus > 18, and use of accessory muscles.⁶⁷ Hypoxemia is common, but CO₂ retention usually occurs only as fatigue and respiratory failure develop.

Chronic Bronchitis. The steady state of chronic obstructive bronchitis is usually associated with some prolongation in the expiratory phase. Scattered coarse rales or rhonchi may be present, often clearing with cough.⁶⁶ The chest may be hyperresonant to percussion and the diaphragms depressed. Exacerbations are associated with wheezing, rhonchi, signs of hyperinflation, and cyanosis, and often with sticky sputum. When the disease is severe, signs of right ventricular failure may be present. These signs include lower extremity edema, neck vein distention, hepatojugular reflux, a right ventricular heave, and a loud pulmonic component of the second heart sound. A tricuspid regurgitant murmur may also be present.

Emphysema. In advanced disease, the patient usually has a hyperresonant chest to percussion and has limited diaphragmatic excursions.⁶⁶ Cyanosis is not common. The respiratory rate is often elevated. During exacerbations or as the disease progresses, tachypnea and tachycardia will be common. Patients often exhibit an upright, leaning forward posture. Spontaneous pursed lip breathing is also common as is the use of accessory muscles. Pursed lip breathing causes a fall in respiratory rate and an increase in tidal volume leading to a reduction in total minute ventilation, a fall in P_aCO₂, and a rise in P_aO₂.⁶⁸ Paradoxical motion of the abdomen is frequent with advanced disease. Right ventricular failure is, however, unusual in the predominantly emphysematous disease states.

Evaluation. Overlap of signs and symptoms of asthma, asthmatic bronchitis, chronic bronchitis, and emphysema may make a specific diagnosis difficult in the absence of measures of lung function such as the FEV₁ and the FEV₁/FVC

ratio. Early disease may be particularly difficult to detect on physical examination and requires spirometry for improved detection. At times, more detailed testing involving assessment of residual volume or the diffusing capacity for carbon monoxide may be needed for a more complete evaluation. Concurrent cardiac disease may also be difficult to distinguish from signs and symptoms of COPD and, in appropriate cases, may require additional evaluations. A comprehensive history and physical examination, however, still offer an excellent basis for both diagnosis and assessment of physiology as a basis for treatment.

Spirometry

The first step in evaluating overall lung health is spirometry, a test that measures the volume of air expelled from fully inflated lungs over time. Spirometry is simple and inexpensive to conduct, and is highly reproducible for an individual. It has been shown to correlate with morbidity and to predict life expectancy, including deaths from all causes. If abnormalities are found, spirometry may be helpful in defining the nature and severity of the ventilatory defect, although further testing may be warranted for complete definition of the other physiologic abnormalities that may coexist with abnormalities in ventilating function, that is, lung volume and gas transfer (diffusion) abnormalities. The reproducibility of spirometry makes it a good test with which to follow lung health over time and to assess the efficacy of therapeutic interventions.

Spirometry is performed by having the patient exhale as fast and hard as possible from a maximal inhalation, while obtaining a trace of the expired volume against time. Most pulmonary testing laboratories may include an inspiratory portion in the test. Although many indexes, both inspiratory and expiratory, may be derived from the spirometric tracing, it is the indexes of forced expiration that are most commonly used to assess lung function. A time-volume curve of a forced expiration is shown in Figure 5 (*top*). The total volume forcibly exhaled is the FVC. The amount exhaled in the first second is the FEV₁. Both measurements are expressed in liters.

Spirometry may also be graphed as flow-volume curves, in which flow (the derivative of volume) is recorded against simultaneous volume (Fig 5, *bottom*). The flow rates are highest in the beginning of exhalation and then fall as lung volume decreases. The data contained in the time-volume and flow-volume curves are exactly the same and may be recorded simultaneously, but the different presentations of the information allow quick visual recognition of various abnormalities in expiratory airflow.

The characteristic pattern of spirometry curves is thought to be determined by the elastic recoil of the lung tissue and the resistance of the airways. Therefore, in diseases of airflow obstruction, for example, asthma, the air empties more slowly, giving the characteristic obstructive spirogram (Fig

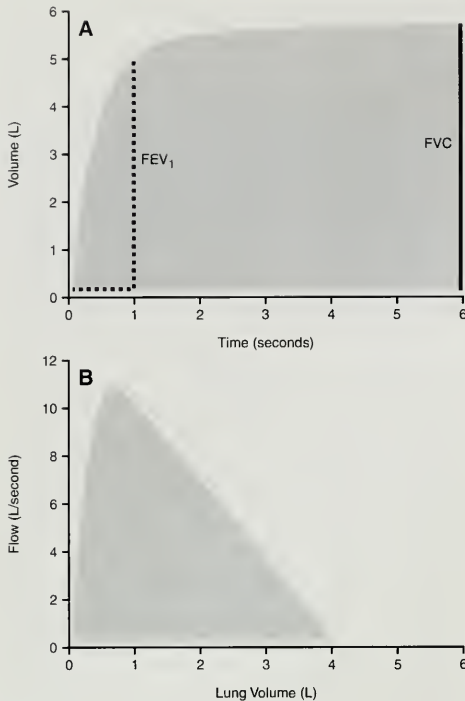


Fig. 5. Normal forced expiration curves. (5A) Volume-time spirogram in which volume the patient exhales in liters is plotted against time. Two common indices of expiratory function, FVC (forced vital capacity) and FEV_1 (forced expiratory volume in the first second), are shown on the trace. (5B) Flow-volume curve may be obtained at the same time as a volume-time curve, but plots expiratory flow in L/second against lung volume in liters.

6, top). In a disease of lung tissue stiffening, as might occur with fibrosis or heart failure, the lung volumes are smaller, although the rate of lung emptying is normal for that smaller volume, giving the characteristic restrictive flow-volume curve (Fig 6, bottom). In a disease like emphysema, two factors, collapsible airways and decreased elastic recoil, contribute to the obstructive spirometric picture. The ratio of the FEV_1 to FVC is normally 75 to 85%, depending on the patient's age.

The usefulness and accuracy of spirometry depend on good quality assessment, starting with equipment selection, equipment validation, and proper test performance technique that includes appropriate reference values and correct interpretation and clinical assessment. The importance of each of these steps has led the American Thoracic Society (ATS) to write specific recommendations to develop consistency within and between spirometers.⁶⁹⁻⁷² Spirometry was previously mea-

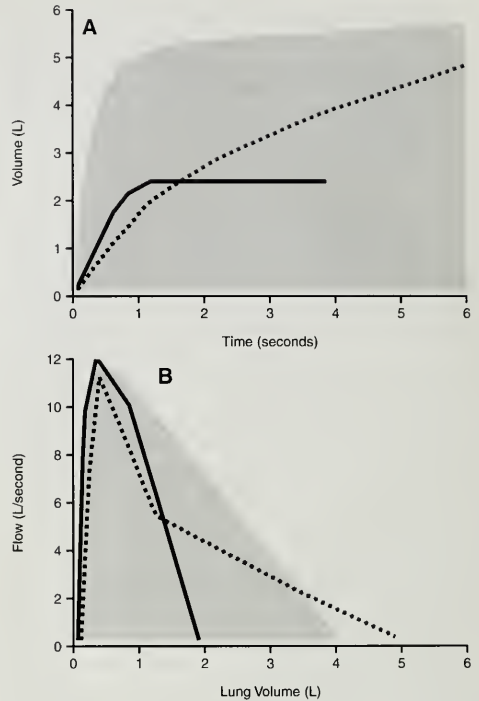


Fig. 6. Abnormal forced expiration curves. (6A) Volume-time traces of a normal pattern (shaded area), a restrictive pattern (—), and an obstructive pattern (---) are shown on the same graph. (6B) Flow-volume curves showing the same three patterns as in (A) also superimposed on the same graph.

sured with a bell-type water seal spirometer requiring calibration and temperature correction of the recorded volumes. While this type of equipment may still be appropriate for a formal pulmonary function laboratory, many accurate and inexpensive electronic spirometers with built-in calibration, reference values, and appropriate temperature corrections are now available and adequate for screening purposes. All equipment should come with a validation by the manufacturer and other independent laboratories that it meets ATS performance criteria.

New recommendations for the performance of simple, handheld spirometers for ambulatory testing and monitoring are being developed. Costs of practical office devices should be modest. Once purchased, the equipment should be checked routinely and calibrated with a large syringe that is provided by the manufacturer. The transmission of infections of all types is easily prevented with proper disinfection and sterilization techniques, proper use of barrier devices like gloves and fil-

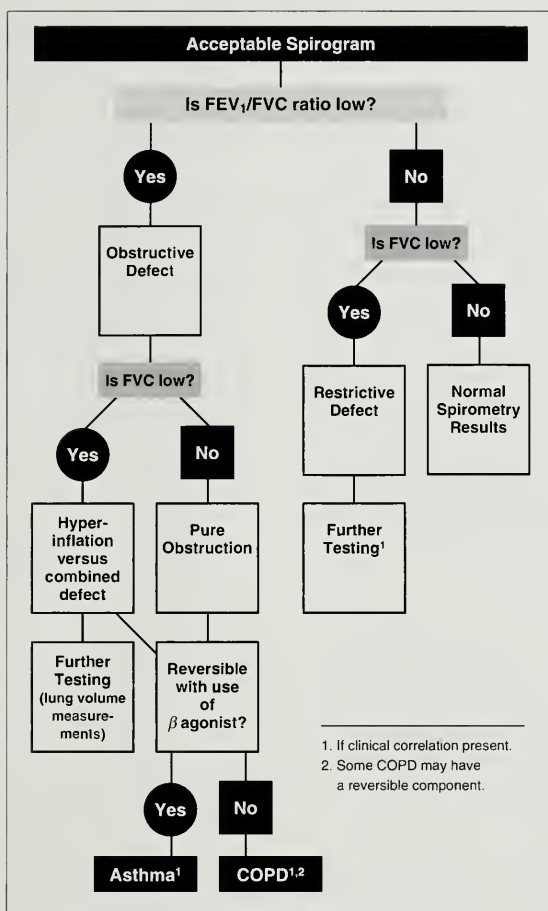


Fig. 7. Algorithm for interpreting spirometry results.

ters, and disposable mouthpieces. With disposable transducers for sensing and recording of only expiratory airflow, contamination is avoided since there is no inspiration with the simple hand-held device. The disposable flow transducers touch only the patient's mouth so there is no risk of inhaling infectious contaminants.

Although simple to learn, spirometry is an effort-dependent test that requires a cooperative patient and an experienced person administering the test. The interaction between the patient and the test administrator is crucial in obtaining the best possible results. The right encouragement from the test administrator to continue exhaling for the required minimum of 6 seconds or until a plateau has been reached may make

the difference between normal and abnormal results. A sufficient number of test maneuvers of good quality must be performed to verify reproducibility. Good reproducibility of the spirometry data is a sign of good test quality. If tests are performed using the same equipment, correction of the raw test data for temperature and daily local barometric pressure will not be necessary to follow the patient over time. Several normal standards are available for comparison with patient data.⁶⁹ Test results must be compared with normal standards that are appropriate to the population being tested. The "normal" value for any patient will vary with age, gender, height, and race. Finally, correct interpretation of the results depends on clinical assessment and correlation. Figure 7 offers an interpre-

tation algorithm that distinguishes obstructive from restrictive ventilatory abnormalities.

Spirometry gives a simple expression of a complex process. Spirometry is an integrated test that may detect pulmonary as well as cardiac or neuromuscular diseases. If abnormalities in screening spirometry are found, referral to a full pulmonary function laboratory for further tests, such as response to bronchodilators, absolute lung volumes, diffusing capacity, or inspiratory force, may help define the differential diagnosis and direct further diagnostic testing. Pulmonary consultation for assistance in interpreting the results of full pulmonary function testing may be appropriate at this time.

Spirometry, like the measurement of BP, provides a useful, simple, and inexpensive assessment of lung as well as general health and is a predictor of morbidity and mortality. Spirometry can be used to follow lung health over time, to reinforce the benefits of smoking cessation, and to evaluate the effects of bronchodilators and anti-inflammatory drugs. As with other screening tests, abnormalities detected by spirometry need clinical assessment and may require further testing. Quality assurance of accurate clinical spirometry is essential to avoid misdiagnosis or abuse of this critical identification and monitoring device.

Chest Radiographs

Radiographic examination of the chest is an important test for the evaluation and diagnosis of COPD.⁷³⁻⁷⁹ In the early stages of these diseases, diagnosis by chest radiograph alone cannot be made with any accuracy. But as the diseases progress, the anatomic changes that occur as a result can be seen and evaluated by chest radiographs. For the sake of clarity, the radiographic appearance of chronic bronchitis and emphysema will be discussed separately even though in many clinical instances these two coexist.

Chronic Bronchitis. In the initial stages of the diseases, the radiograph of the chest can be completely normal in patients suffering from COPD. Increased lung markings sometimes referred to as "dirty chest" are a common manifestation of chronic bronchitis. This is ascribed to chronic inflammatory changes and mild perivascular and peribronchial fibrosis, and thickening of the bronchial walls. Often, especially in heavy smokers, medium-sized bronchi can be seen on end in the hilar area. These bronchi show increased intimal thickening, reduction of the lumen, and sometimes irregularity of the bronchial walls. Occasionally, thickened bronchi can be seen longitudinally as air-containing tubular shadows. This appearance is often referred to as "tram lines." The inner walls of these bronchi can appear swollen and irregular.

Emphysema. The presence of advanced emphysema in COPD patients results in distinct radiographic findings. The hallmarks of emphysema are hyperinflation of the lungs, flattening of

the diaphragms, changes in pulmonary vasculature resulting in oligemia, and the presence of different types of bullae. Hyperinflation of the lung is manifested in the chest radiograph as depressed diaphragms, anterior bowing of the sternum, increase in the retrosternal and retrocardiac spaces, narrowing of the mediastinum, and decrease in the size of the heart and the vascular pedicle width. Reduction in the caliber of pulmonary arteries with tapering of the vessels at the periphery is often seen in the oligemic lung. Other changes in the vasculature include sudden cutoff of side branches and sometimes their obliteration, with splaying of the angles of bifurcation of the blood vessels, and displacement of the vessels adjacent to emphysematous bullae. Pulmonary hypertension as a result of COPD is manifested as an increase in the size of the hilar markings and an increase in the diameter of the right pulmonary artery in excess of 16 mm. In patients with chronic bronchitis and emphysema, there is a regional and diffuse attenuation of blood vessels with loss of side branches and increased definition of vessel walls. Emphysema also causes a loss in the alveolar pattern or the alveolar flush causing the lung to appear dark in the plain radiograph. Emphysema causes a widening in the peripheral clear zone that may reach as far as the middle of the lung fields.

In COPD with emphysema, the mediastinum is usually long and narrow with a diminished size of the heart and the vascular pedicle width. When left heart failure is present, as is common in COPD, the size of the heart becomes normal and the signs of hyperinflation disappear. There is increased congestion of blood vessels where the vasculature is normal, and the focal areas of emphysema become more evident, giving rise to the "Swiss cheese" appearance. The presence of bullae is common in emphysema. They appear as large air-containing cystic areas of varying size within the lungs. They are thin walled and can be single or multiple. Large bullous formations are seen at the bases in panacinar emphysema, ie, emphysema that is uniformly present through the acinus. Bullae can occur without anatomic presence of emphysema as in the congenital condition sometimes referred to as paraseptal emphysema. There is some evidence that patients who show the arterial deficiency pattern in the chest radiograph have panacinar emphysema and that those who show increased markings and congestive pattern have centrilobular emphysema, ie, loss of alveolar walls within the center of the acinus. The panlobular form of emphysema exists throughout the lungs and is most often associated with α -1-antitrypsin deficiency. The centrilobular patterns of disease are closely related to smoking and are most prominent in the upper lung zones. Most patients who show the increased markings pattern also have attendant pulmonary hypertension, cor pulmonale, and right ventricular enlargement.

Ventilation perfusion studies using xenon have shown abnormalities that have a correlation with the existence of COPD, and they are especially helpful during the stages when pulmonary function test results may be normal. Computed

tomography (CT) scans of the chest have been found to be accurate in identifying patients with the various patterns and distribution of emphysema. They are useful in assessing the extent of the disease and in estimating the size and the number of bullae and the location of the greatest degree of distribution, which is most commonly in upper lung zones. This information is of special value in the evaluation of patients for lung volume reduction surgery. Radiographic examination of the chest in COPD patients has the added benefit of uncovering coexisting diseases such as unexpected cancer of the lung, heart diseases, and infections. It is important to remember that signs of hyperinflation are also present in patients with acute attacks of asthma without any evidence of COPD. In asthma, however, the appearance of the blood vessels is within normal limits, and bullae are not usually found.

It is recommended that physicians obtain chest radiographs in patients with COPD to look for the presence of coexisting conditions such as lung cancer, cor pulmonale, pneumonia, pneumothorax, heart failure, and bullous disease, and to assess the overall severity of the disease. In summary, radiography of the chest is not a screening test for the diagnosis of COPD. However, in the late stages, a reasonably accurate diagnosis of COPD and emphysema can be made from the chest radiograph and CT scan.

8. Prevention

Prevention is the goal of all of medicine. This includes both primary and secondary prevention. Of course, it would be ideal to prevent COPD. The factors that impact on prevention are basically three.

The first is smoking, which is by far the greatest risk factor in the causation of COPD. Right now we are not making much progress in preventing teenagers from starting to smoke. This is because of the advertising success of the tobacco industry that daily seduces 3,000 teenagers into the high risk of nicotine addiction. This number equals the sum of those who die each year of tobacco-related diseases or who finally quit smoking. Thus, we are at a dynamic equilibrium. It is a sorry fact that more teenagers than adults older than 20 years of age smoke cigarettes today!

The second controllable risk factor is occupational exposures. Those who work in dusty environments are most vulnerable, such as grain workers. However, a change in occupation and a clean environment is not possible for many people.

The third risk is hereditary susceptibility. An α -1-antitrypsin deficiency state is responsible for only a small number of COPD patients, probably 3 to 5%. Replacement therapy is expensive and its value not yet proven. Constitutional susceptibility factors account for many other genetically determined forms of COPD and are not changeable.

The identification of and intervention in early stages of asymptomatic COPD are the basis of secondary prevention. Smoking cessation and possibly the use of anti-inflammatory

agents such as antioxidants and antiproteases offer the potential for secondary prevention. Antimicrobial vaccines against influenzae and the pneumococcus should be used.

The use of pulmonary rehabilitation techniques and the treatment of acute respiratory failure can be considered within the realm of secondary prevention. The goal is to prevent and forestall premature morbidity and mortality.

Of the 30 million or so people with various stages of COPD, only about 50% are symptomatic. The other 50% are already on the pathway to potentially disabling COPD, but neither the patient nor the physician may be aware of this serious health menace. Future efforts of the NLHEP will apply emerging strategies for both primary and secondary prevention. This section offers a contemporary commentary on the approach to prevention and how we may be able to change the course of COPD, but this depends on the patient and the physician becoming more aware of this serious health problem.

The statistics presented in Section 5 clearly point out the need to develop, promote, and emphasize prevention in COPD. Currently, our understanding of prevention in COPD encompasses mainly secondary preventive approaches such as smoking cessation, screening for early recognition, diagnosis, and early management; and tertiary prevention such as respiratory rehabilitation programs. Primary prevention for COPD, at this time, is reduced to smoking prevention. This section will cover all of the above, starting with spirometric screening.

Screening for Early Diagnosis

Historically, public health efforts have centered on mass screening models for many chronic diseases: a baseline mammogram is recommended for all women older than 40 years to screen for breast cancer and all men older than 50 years for prostate cancer. Screening by prostate-specific antigen and digital rectal examination may identify asymptomatic stages and this screening is recommended by the American Cancer Society. Colonoscopies are recommended for those at risk for colon cancer. The "know your numbers" campaign for cholesterol screening has become ubiquitous.⁸⁰ Screening for diabetes is commonly performed, and BP measurement is routine during all health care visits. In each case, it took several years for the process to become accepted and widely applied.⁸¹ Of the accepted screening procedures mentioned above, only BP screening is noninvasive. Similarly, spirometry is also noninvasive, requiring < 5 minutes to complete by trained and qualified individuals.¹¹ Before they became accepted, most screening procedures described lacked data. This was previously the case with spirometric screening for lung health. Data regarding both the cost/benefit ratio for various at-risk populations, as well as data on the potential misinterpretation of results are needed as well. One argument is that this lack of data should not be a deterrent to examine the potential advantages and disadvantages of such an approach. How would the

results of screening for lung health translate into public health directives, ie, prevention recommendations? Would the costs of screening for lung health be offset by its potential benefits? Would widespread use of spirometric screening provide some answers as was the case in many of the examples above? After all, guidelines for mammography are still being revised as experience gained clarifies our understanding. Consider the fact that there have been no randomized controlled clinical trials to prove the cost-benefit value of the pelvic Papanicolaou test in women at risk of cervical or uterine cancer. The dramatic reduction in fatality from both cervical and uterine cancer answers the question without a trial and without a doubt!

The results from the Lung Health Study show that COPD is nearly as widespread in women as men, and that it is far more common in young individuals than was thought.⁸² Another finding of the Lung Health Study is that a subgroup of smokers was shown to be especially sensitive, losing lung capacity at a much faster rate than other less sensitive persons.¹ Spirometric measurements for such an "at risk" group may be a start, followed by a more aggressive referral and follow-up procedure for this group to attend smoking cessation programs; also, coupling these "recommendations" with incentives such as reduced health care costs as they continue to abstain from smoking may be a fruitful approach (similar to many insurance company policies).

To yield the greatest understanding of lung health, clinical spirometry should become as ubiquitous as measuring BP. In addition to becoming part of all health care visits, spirometric screening for lung health should take place at the workplace where occupational exposures, eg, organic dusts, are known to constitute risk factors for COPD.^{83,84} Spirometry in the workplace should be coupled with wellness programs, encouraging the use of protective equipment, smoking cessation programs, and reducing the levels of ambient irritants. Until the data regarding the role of environmental indoor and outdoor pollutants are more specific, few public health measures other than regulatory can be instituted.⁸⁵

Smoking Prevention

Several interested organizations suggest that smoking prevention may be an avenue more promising than smoking cessation. The National Academy of Sciences' Institute of Medicine, in its publication *Growing Up Tobacco Free*, makes a good case for "A Youth-Centered Prevention Policy" where a review of "School-Based Smoking Prevention Programs" showed them to be especially successful when peer led, reducing by 35 to 50% the daily and weekly smoking incidence over adult-led programs. However, as with other behavior modification approaches, booster sessions are important.⁸⁶ The Public Health Service, in its second "Healthy People 2000" review on tobacco, stated that a main objective is to reduce the initiation of cigarette smoking by children and youth so that no more than 15% become regular smokers by age 20

years, stressing the need to develop key elements of the Public Health Service strategic plan to focus on youth.⁸⁷ The Food and Drug Administration has called childhood addiction to nicotine "a pediatric disease" since the average smoker starts at age 14 and a half years and becomes a daily smoker before age 18 years.⁸⁸ Data from the National Health Interview Surveys show that based on a median initiation age of 16 to 17 years, 50% of adult men will have already smoked at least 16 years and women 20 years.⁸⁹ The American Lung Association (ALA) and American Thoracic Society (ATS) make a point to target smoking prevention by citing that school-based programs have been shown to delay the onset of smoking by 2 to 4 years, a significant effect in light of the above statistics.⁹⁰ In fact, the ALA/ATS dedicated the opening session of their annual meeting in 1996 to "the issue of tobacco use as a 'pediatric disease.'"⁹¹

Cost-Benefit and Cost-Effectiveness Analyses

To proceed with realistic recommendations, cost-benefit and cost-effectiveness data analyses dealing with prevention of COPD are needed. Several studies dealing mainly with the cost comparison of various treatment choices have been identified;⁹⁰ however, in the area of prevention of COPD, those detailed below were found in relation to prevention of the progress of asymptomatic disease and in the treatment of symptomatic patients. The cost-benefit analysis of the use of bronchodilators in COPD showed that maintenance management with ipratropium bromide was less costly and more effective in reducing complications than maintenance management with theophylline.⁹²

Preventive Approaches

Some preventive approaches such as influenza vaccinations have been shown to be cost-effective⁹³⁻⁹⁵ even in nonpandemic years. These findings led McBean et al⁹⁵ (1993) to conclude that, "There is the possibility of large cost savings to both Medicare and the population in general if effective influenza control strategies are implemented." Other approaches, such as replacement therapy for α -1-protease inhibitor and the pneumococcal vaccine, are still undergoing evaluation. One study on the cost-effectiveness of smoking cessation in Wales looked at a 4-year period (1984 to 1989) in terms of morbidity and "displaced mortality." The conclusions are that large-scale benefits to the national economy can be derived from reductions in smoking.⁹⁶

Rehabilitation

Pulmonary rehabilitation can be considered as preventive care designed to prevent or forestall premature morbidity and mortality in the more advanced, symptomatic stages of disease. The increased costs of institutionalization have spawned

the home health care industry. A few home care programs for patients with COPD have been evaluated and found to be cost-effective. The outcome measures of interest include the following: improvement in quality of life, dyspnea, and the 6-minute walk, as well as the more traditional ones, ie, number of hospitalizations, length of stay, and number of emergency treatments. A growing body of data shows that pulmonary rehabilitation programs that combine education with exercises and counseling improve the above outcomes along with social and physical functioning, resulting in a decrease in symptoms of depression. These measures, in turn, translate into reduced morbidity and thus into economic savings.⁹⁷⁻¹⁰²

Exercise Training. Exercise conditioning is important to increase the efficiency of the muscles in extracting oxygen from the blood delivered, thus reducing the ventilation and heart rate needed for a given workload. It can improve the sense of well-being and is as important in persons with airflow obstruction as in trained athletes who have been sidelined due to injury or other circumstance. This is an important concept and should be presented to patients as a vital part of the educational program.

Exercise training is largely task specific. Thus, the program must include systematic exercises of muscles that the patient will use in everyday activities, such as walking and arm motion. Upper arm training is very important since arm activity can produce unusual dyspnea because of competition with accessory respiratory muscles, among other factors. In asymptomatic patients with early airflow obstruction, simple verbal instruction for exercise will suffice. However, when COPD causes exercise limitation, referral to a respiratory specialist who has access to a pulmonary rehabilitation program may be advisable (see Section 12). This type of program defines the limits of the patient's exercise tolerance, including the amount of conditioning needed, and creates an individual exercise "prescription." The specific training of respiratory muscles may additionally improve exercise and breathing comfort.¹⁰² Maximizing the patient's participation in prized everyday activities is most likely to enhance quality of life. The better the patient is educated in these matters the more successful will be the partnership in maintaining good lung function and the participation in a therapeutic strategy designed to mitigate the physiologic and psychosocial impact of COPD.¹⁰³

One study calculated the cost-benefit ratios of educating patients with asthma compared to patients with COPD in terms of quality of life and the usual morbidity measures (hospitalizations, emergency treatments, and absences from school/ work), and found that whereas the results are favorable in asthma, they are equivocal in COPD.¹⁰⁴ An 11-year review of rehabilitation and home care programs concluded that some evidence favors the use of hospital-based home care programs directed at highly selected, very severely afflicted patient groups, as compared with community-based

home care programs in wide patient populations. The study cited a lack of any cost-effectiveness study concerning pharmacotherapy.¹⁰⁴ Another study showed a greater effectiveness of home-based pulmonary rehabilitation compared with one that was hospital based. The home-based program was far less costly.¹⁰⁵ An important controlled clinical trial has documented improved exercise tolerance and quality of life that were sustained for 1 year.¹⁰⁶ In the future, cost-effectiveness evidence will increasingly be asked for as a complement to the clinical evidence of the safety and efficacy of new drugs. Another important area where cost-benefit data are totally lacking is that of diagnostic technologies. For example, an economic evaluation of spirometry would be of value; similarly, an assessment of the value of screening for α -1-antitrypsin deficiency is needed. The authors point out how it is difficult to compare the results of various cost-effectiveness studies, because the analyses are not conducted according to uniform, accepted methodologic standards.¹⁰⁴

In conclusion, although intuitively attractive and potentially useful, a comprehensive strategy for the prevention of COPD may not be realistic at this time beyond renewed efforts at smoking cessation and smoking prevention, which remain the primary goals. Prevention of the progress of each stage of COPD, including the utilization of spirometric screening, is still in its early stages. More data are needed to answer the remaining basic questions that include concerns about the most practical and useful methods of early identification and intervention in COPD.

9. Physician-Patient Partnership through Education and Management

Education

Healthy living practices provide the most effective method of preventing COPD. Avoidance of smoking is probably the single most important behavior to protect the lungs throughout life. This concept should be instilled as early in life as possible, through family, school, and the media, as well as the primary care practitioner at the time of an individual's routine health examination. The partnership of each person with his or her educators and health care providers is important in maintaining lung health and preventing pulmonary disease and other diseases found to be predicted by the presence of airflow limitation. The total alveolar surface of adult lungs, which exceeds 100 m², is exposed to a sometimes hostile environment. It is clear, then, that community and public health efforts to improve ambient and personal air quality offer great general benefit. If early airflow obstruction is detected in a periodic health check, every effort should be made to prevent accelerated worsening of maximum airflow in such patients. It is essential to educate the patient and his or her family about the consequences of accelerated airflow obstruction (that is, decline of FEV₁) over time, if action is not taken. Such edu-

cation can be carried out by the primary care physician or a specialist health care provider to patients individually or in groups.¹⁰⁷ A partnership of all concerned benefits the patient's future. Even though the patient, in whom a moderate reduction in FEV₁ is incidentally detected, is asymptomatic, this is the optimum time to change the abnormal annual decline in FEV₁, and thus attempt to prevent a number of lung and cardiovascular diseases from developing.

Smoking Cessation. Most smokers who successfully stop smoking do so on their own, even though their efforts may be met with many relapses before they achieve permanent success at "clearing the air." Once early airflow obstruction is detected in a smoker who needs help in stopping, a program of intervention becomes crucial. It is important to remember that smokers want help from their physicians and other health care professionals. A strong message by the health care provider is the essential first step in smoking cessation, and it should focus on educating the patient about normal lung function and the natural history of smoking-related diseases.¹⁰⁸ Some physicians or smoking interventionists ask that the patient sign a contract that formalizes the planned effort to stop. Setting a date to stop smoking abruptly has consistently proved more effective than gradually tapering the amount of smoking. Setting a firm quit date allows for anticipation and attitude adjustment toward smoking.

Many reinforcement techniques can be used to assist smokers. They need to be reassured that the likely moderate weight gain is a lesser evil than continued smoking and that the problem can be ameliorated by a regular exercise program started even before stopping smoking. Education concerning wise choices in the amounts and types of food is also an important feature of a successful smoking cessation program. Repeated reinforcement has been shown to be necessary to achieve a higher rate of compliance.¹⁰⁹ Whenever follow-up by the health professional ceases, the chance of relapse increases. Printed self-help material is readily available to help the smoker prepare to quit and reinforce the decision.¹¹⁰ Formal support groups and the buddy system are especially important during the first few months of smoking cessation. Group support programs and printed self-help materials are available in many communities through national lung and cancer societies, local hospitals, and church groups. These programs have been relatively successful and many more need to be established with the assistance of various volunteer health groups.

Pharmacotherapy is also available for those who need help beyond behavioral motivation. Nicotine gum, now available over the counter in 2-mg and 4-mg strengths, has been shown to improve the smoking cessation rate when added to behavioral and motivational methods in appropriate candidates.^{111,112} The rationale for this approach is to stop the activities associated with smoking while still maintaining a blood and tissue level at about half that of a pack-a-day smoker. After some weeks or months, depending on the individual, the substitute

nicotine source helping to ease the addictive features can be gradually decreased or abruptly stopped.

Nicotine patches provide additional convenience and have different and possibly fewer undesirable effects than non-pharmacologically aided smoking cessation. Nicotine patches double the quit rate achievable by various levels of behavioral modification used alone.¹¹³ The newly released nicotine nasal spray adds to our armamentarium as an additional strategy to help mitigate nicotine withdrawal symptoms in the addicted smoker.¹¹⁴ Ongoing research into approaches with psychoactive drugs to help reduce the addictive craving of cigarettes is underway. Detailed instructions and repeated reinforcement in proper use are equally important in pharmacotherapy as well as in all aspects of management to ensure compliance and to avoid relapse. Nasal nicotine spray is now available by prescription. It should be used in the most "hard core" smokers.¹¹⁴ The antidepressant bupropion (Zyban) is also as effective as the nicotine patch in smoking cessation. When compared with placebo, patients who received 200 or 300 mg of bupropion per day had a quit rate at 1 year of 22.9% and 23.1%, respectively, proven by carbon monoxide testing.¹¹⁵ A summary of pharmacologic agents approved for the treatment of nicotine addiction is presented in Table 3.

Table 3. The Summary of the Pharmacologic Agents Indicated for Assistance in Smoking Cessation

Method of Administering	Unit Dose	Dose Interval
Oral: nicotine polacrifex	2 mg 4 mg	Every 1-2 hours Every 1-2 hours
Bupropion sustained-release tablets	150 mg	150 mg for 3 days; then 300 mg each day (2 tablets)
Patch: transdermal nicotine (3 types)	21 mg, 14 mg, 7 mg 15 mg, 10mg, 5mg 22 mg, 11mg	Over 24 hours Over 16 hours Over 24 hours
Spray: nasal nicotine	0.5 mg/inhalation/ nostril	8-40 mg per day in hourly or PRN* dosing

*PRN = as occasion requires.

10. Clinical Management

Many management options are available for the clinician to relieve symptoms and improve quality of life in patients with all stages of symptomatic COPD. Only oxygen has been the subject of randomized controlled clinical trials, which showed it to increase survival and improve quality of life. In addition to oxygen, there are other strategies, including vaccines and other pharmacologic measures, recognized by clinicians to be valuable management tools for COPD. The benefits and limits of each of these are discussed in the following pages.

Vaccines

Patients with COPD have an increased risk for respiratory tract infections. Infection prophylaxis via vaccination can reduce the incidence and severity of bronchial infections, reducing the morbidity and mortality associated with these infections.¹¹⁶ Annual vaccine prophylaxis against influenza is recommended for all COPD patients, with the vaccine formulation and potency revised yearly.^{11,116} Chemoprophylaxis with amantadine and similar agents should be considered when unimmunized patients are at high risk for contracting influenza and there is inadequate time for immunization to become effective or immunization is contraindicated, which would be the case with a prior allergic reaction to influenza vaccine or a known allergy to eggs. Amantadine can be given as a single daily dose of 200 mg during periods of high risk of influenza.¹¹⁷

Although the benefits of the pneumococcal vaccine have been debated, pneumococcal vaccination is currently recommended for all COPD patients, with revaccination to be considered every 6 years in those patients at high risk for significant reductions in immune function.¹¹⁶⁻¹²⁰ Routine revaccination is not recommended for all patients due to uncertainty over the rate of decline in immune response and concern about immune reactions in immunocompetent patients. The current pneumococcal vaccine is 23 valent, covers > 80% of pneumococcal strains, and is 60% effective in producing antibodies in immunocompetent patients. Both influenza and pneumococcal vaccines are underutilized. Less than 30% of high-risk patients are immunized for influenza each year and < 10% of patients receive the pneumococcal vaccine.¹¹⁹

Bronchoactive Drugs

The judicious use of bronchodilator medications can improve function in patients with COPD. Prior to initiating any medical management regimen, each patient should undergo a baseline evaluation, including a careful history and physical examination, laboratory tests as indicated by the history and physical, chest radiograph, and spirometric testing, before and after bronchodilator administration. Unfortunately, a single trial of bronchodilators in an individual patient does not accurately identify patients with partially reversible disease, nor does it predict the response to long-term bronchodilator therapy. Thus, the systematic use of bronchodilators in a stepwise fashion is recommended, a method that can provide significant increases in expiratory airflows and symptomatic relief, even in patients with "fixed" airflow obstruction.¹⁰⁷ Although a stepwise approach is warranted, it must be recognized that individual patients have specific medication intolerances and contraindications that require customizing of therapy. A summary of the stepwise approach to bronchoactive pharmacologic therapy is presented in Figure 8.

Bronchodilators can be given by inhaled, oral, subcutaneous, and IV routes. However, the inhaled route is preferred

due to the direct effects on the lung with relatively less systemic effects. Use of a metered-dose inhaler (MDI) with or without a spacer device provides a response equal to that of nebulized therapy. Delivery of inhaled bronchodilators by intermittent positive pressure breathing offers no additional advantage. When used properly, MDIs (if need be, with a spacer) greatly simplify inhaled therapy, allowing for improved patient compliance and decreased equipment costs and care. If dyspnea and severe bronchospasm during exacerbations impair MDI technique, nebulizer therapy may be necessary. Whatever therapy is initiated, it is imperative to monitor patient symptoms, expiratory airflow, ie, FEV₁, exercise tolerance, medication usage as occasion requires, and need for medical attention to determine patient response to treatment.

The most common mistakes in bronchodilator management include inadequate education regarding medications and inhaler techniques, suboptimal medication dosing (too little or too much), inadequate monitoring of patient response to treatment, and a lack of pretreatment prior to activity. Patient education should include a rationale for medical treatment and specific education related to medication dosing and timing and the proper use of the various inhalation devices. Instructional aids include nationally and locally produced self-instruction written materials and audiovisual aids. In addition to these, individual training is frequently needed during initial MDI and spacer education. Repetitive instruction of proper MDI technique is mandatory and patients should be required to demonstrate mastery of these skills over the course of their care. Family members should be included in the education process, as their participation in the patient's subsequent care is crucial both to success and to perception of success. Anticipated outcomes from this education include more appropriate medication usage and increased compliance.

Anticholinergics

Anticholinergic agents are now an integral part of COPD therapy and are considered to be first-line agents by many.¹⁰⁷ Anticholinergics such as atropine and ipratropium have been reported to produce greater bronchodilation than β -agonists at conventional dosages, although it has been suggested that maximal doses of either agent result in similar degrees of bronchodilation. Of note, anticholinergics may produce bronchodilation in some COPD patients who have no response to β -agonists. Ipratropium has an advantage over atropine in that it is poorly absorbed into the circulation and does not cross the blood-brain barrier, resulting in minimal side effects, even when used in very high dosages. The slower onset of action and longer duration of action of ipratropium may make it more suitable for use on a regular basis. In addition, ipratropium has been shown to reduce sputum volume without altering sputum viscosity. The recommended dosing for ipratropium (2 puffs qid) is suboptimal in many patients and higher dosages (3 to 6 puffs qid) with a spacer should be con-

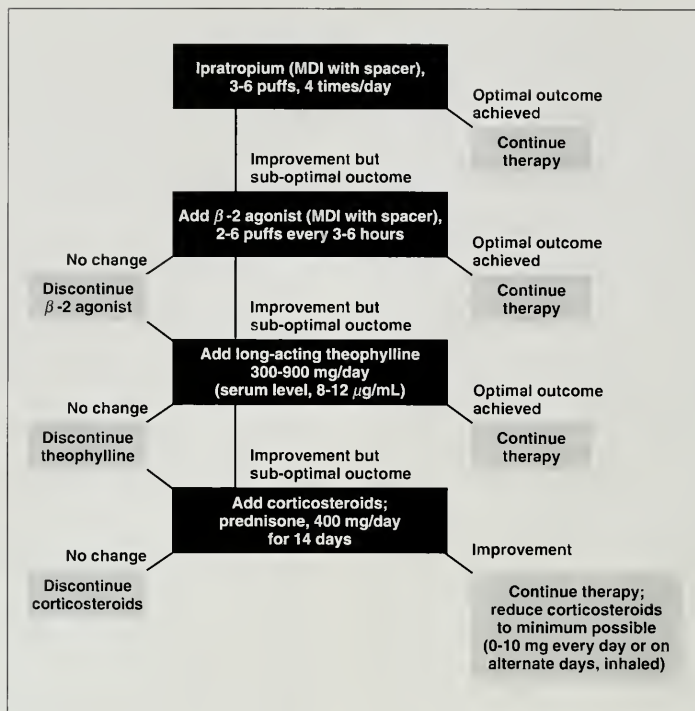


Fig. 8. Typical regimen for treating chronic obstructive pulmonary disease. Outcome was measured in terms of improvement in the forced expiratory volume in the first second (FEV_1), FEV_1/FVC (forced vital capacity), and peak flow; improvement in the distance covered in a 6- or 12-minute walk; and objectively observed reduction in dyspnea, medication use, and nocturnal symptoms. MDI = metered-dose inhaler. From reference 107, with permission.

sidered. The Lung Health Study, which evaluated the regular use of modest doses of inhaled ipratropium early in the course of COPD in an attempt to retard disease progression, showed no beneficial effect on baseline FEV_1 .¹ However, another study did demonstrate an improvement from ipratropium in baseline FEV_1 over a 90-day period compared with albuterol and demonstrated no beneficial effect in baseline spirometric functions.⁵⁶

Sympathomimetics

Sympathomimetics have been the mainstays of COPD management for years, although their role as a first-line agent has been challenged by ipratropium in recent years.^{121,122} Many sympathomimetics are available. There is considerable variation in the time of onset, length of duration, and degree of adrenaline-like kick within the various sympathomimetics based on the degree of α -, β 1-, and β 2-adrenoreceptor ago-

nist selectivity and other intrinsic properties of each agent. β 2-Adrenoreceptor agonists are currently the sympathomimetic agents of choice in the treatment of COPD, minimizing α and β 1 side effects. Nevertheless, the potent β 2-adrenoreceptor agonists can cause tremor and a reflex tachycardia.^{122,123} Most recommended sympathomimetic doses provide less than maximal bronchodilation. Although higher dosing can improve bronchodilation in some patients, tachyphylaxis has been reported and there is a minor concern over the development of a cardiomyopathy and other cardiac toxicities with excessive β 2 agonist usage. The potential for inducing hypokalemia with higher doses of β 2 agonists must also be recognized and monitored in patients at risk. In general, β 2-adrenoreceptor agonists have a rapid time of onset and are preferred for the treatment of acute bronchospasm. Oral β 2 agonists are not recommended at initial therapy because of their high incidence of side effects, but can be tried in patients unable to use inhaled therapies.

Theophylline

Although debate continues about the value of theophylline, its place in the management of COPD suggests that theophylline therapy benefits many COPD patients. The controversy may be due, in part, to misconceptions about the mechanisms of bronchodilation, problems with drug delivery, and evidence that theophylline offers little additional bronchodilation beyond that of inhaled agents. Nevertheless, theophylline therapy has been shown to benefit COPD patients. Evening dosing with long-acting theophylline preparations has been shown to reduce overnight falls in FEV₁ and improve morning respiratory symptoms in patients using inhaled bronchodilators.¹²⁴ Theophylline has also been suggested to have various other respiratory effects, including reduced gas trapping, improved respiratory muscle function and gas exchange, and enhanced mucociliary clearance.¹²³ In addition, theophylline has been shown to improve the central drive to breathe and yet to decrease dyspnea. It also has pulmonary vasodilator effects that can improve right ventricular function and exercise performance in COPD patients.¹²⁵

The toxic reactions of theophylline are well recognized, as are the various conditions that may acutely alter theophylline levels, including hepatic dysfunction and the use of various medications such as cimetidine and erythromycin. If warranted in therapy, theophylline can be given to achieve safe levels in COPD patients in the lower therapeutic range (8 to 12 $\mu\text{g/mL}$). Rarely, higher levels (16 to 20 $\mu\text{g/mL}$) may be of benefit, although close monitoring for side effects and drug toxicity is required. Several long-acting theophylline preparations are currently available that provide stable uniform drug release.

Combined Bronchodilator Therapy

In many instances, the combination of several bronchodilator agents may provide additive effects.^{107,122} The combination of a β agonist and anticholinergic in the same MDI (Combivent) has been shown to have an additive effect, especially when typically recommended (lower) doses of each agent are used.¹²⁶ A combination of albuterol and ipratropium (Combivent) has recently been released in the United States. Simultaneous administration of both agents does not appear to potentiate the bronchodilator effects of either agent. Theophylline and β -2 agonists have also been shown to have additive bronchodilator effects and the combination is usually well tolerated. The combination of several agents may allow for significant bronchodilation while using submaximal doses of each agent, thus helping to avoid drug toxicity.

Corticosteroids

The value of corticosteroids in both stable and acute conditions remains uncertain. Corticosteroids have been shown to produce a favorable response in some COPD patients during acute COPD exacerbations, improving symptoms and

reducing hospital durations. Steroids have also been shown to reduce relapses in a minority of COPD patients with a history of frequent exacerbations. Thus, short courses or bursts of corticosteroids should be considered in COPD patients during acute exacerbations that are unresponsive to aggressive inhaled bronchodilator therapy.

Many COPD patients have corticosteroids indiscriminately prescribed on a long-term basis, producing severe adverse side effects without any lasting positive benefits. In several trials, no more than 20 to 30% of COPD patients showed objective benefits from long-term steroid administration.¹²⁷ To date, it has not been possible to predict which individuals will respond to long-term systemic corticosteroid therapy. Indeed, a beneficial steroid response during an acute COPD exacerbation does not indicate a patient will benefit from long-term steroid use. A closely monitored clinical trial remains the only way to select those patients who might benefit from long-term steroid therapy. A trial of "therapeutic" corticosteroids for about 2 weeks should be considered in patients who have continued symptoms or severe airflow limitation despite maximal therapy with other agents. Only those patients with significant documented physiologic improvement should be considered for long-term therapy, with a goal of reducing dosage to the lowest possible.¹⁰ There is no evidence that long-term alternate day or inhaled steroid usage is beneficial in COPD patients, although this seems logical in patients with a clear asthmatic component, and selected patients should be treated in this fashion. Short-term studies of high-dose inhaled steroid in COPD patients do suggest there may be some benefit to these agents, but criteria for identifying appropriate patients have not been established.¹¹ If inhaled steroids are utilized in maintenance management, their ability to sustain a continued steroid benefit needs to be documented by serial spirometric measurements. Some support for the use of inhaled corticosteroids, along with inhaled β agonists, is accumulating.^{53,55,128}

Regular long-term use of low-dose oral corticosteroids has been suggested to retard the rate of decline in FEV₁ in COPD patients.¹²⁹ Studies evaluating the regular early use of oral and inhaled corticosteroids as a means of retarding disease progression are ongoing. Until this information is available, the use of steroids solely to retard the progression of disease cannot be recommended. It should be recognized that the optimistic adding of steroids to the drug regimen in COPD may add to the burden of therapy and result in decreased compliance for all the medications. Thus, long-term steroid therapy should be added only when benefit can be shown and the patient's compliance can be ensured.

Strategic Drugs

Mucolytics. Although clearance of thick, tenacious secretions can be a major problem in COPD patients, there is little evidence that thinning of secretions or improving sputum

clearance rates produces any clinical improvement.¹¹ Increasing fluid intake is of no value unless, possibly, in those patients who are dehydrated. Nebulizing water or hypertonic saline solution is without documented benefit and may irritate the airways and induce bronchospasm. Oral expectorants such as guaifenesin and iodides have not been shown to be of benefit in most COPD patients. Guaifenesin is frequently prescribed as a mucocactive drug in the United States. One controlled clinical trial failed to prove efficacy.¹³⁰ Iodinated glycerol has been suggested to improve cough and chest discomfort in some patients with chronic bronchitis, but this preparation is no longer marketed.¹³¹

Of the many mucolytics, acetylcysteine has had the widest use and has been shown to thin secretions of patients with chronic bronchitis.¹³² However, it has no effect on the volume of sputum produced or on airflow in patients with COPD, and the aerosolized drug can induce significant bronchoconstriction. Interestingly, acetylcysteine has been shown to have antioxidant properties that may be of benefit to COPD patients, and the oral drug that is marketed in many countries is often used in standard therapy. Various other mucolytic agents for COPD have also been suggested, including pancreatic dornase, surfactant and various proteolytic agents, and detergents. These agents are not generally available, and cannot be recommended since they are of unproven value in the treatment of COPD patients. Cough suppressants should be avoided or prescribed cautiously, since they may impair secretion clearance and could theoretically increase retained secretions. There is no evidence that they enhance the risk for respiratory tract infections.

Selected COPD patients with excessive secretions or an ineffective cough may benefit from postural drainage and chest percussion. However, chest physiotherapy is not of benefit to most patients with COPD, although vibration at appropriate frequency can loosen secretions.^{133,134} Various other physical methods, including positive expiratory pressure, forced expirations,¹³⁵ and even a nonproductive cough may help mobilize secretions.^{135,136} Flutter valve therapy has recently been suggested to facilitate airway secretion clearance.¹³⁷ There is emerging information documenting the efficacy of these devices in COPD.

Reductions in mucus hypersecretion may be of equal importance to enhanced secretion clearance. Avoidance of airway irritants, including cigarette smoke, occupational dusts, and high air pollution, and treatment of significant postnasal drainage, gastroesophageal reflux, and infections may reduce airway secretions. Some of the macrolide antibiotics have also been suggested to decrease mucus secretion, without altering other mucus properties.

Antibiotics. Although still somewhat controversial, antibiotics are frequently used in the treatment of COPD exacerbations and have been shown to shorten the course of acute exacerbations.^{138,139} Since most infectious exacerbations are viral in origin, it is not surprising that in typical cases, antibi-

otics are of dubious benefit. Examination of sputum Gram stains for purulence may be of help in determining the need for antibiotics. Sputum cultures are not cost-effective unless there is evidence suggestive of concomitant pneumonia. In most situations, broad-spectrum antibiotics capable of treating *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis* are preferred. Suitable agents include ampicillin, amoxicillin, cephalosporins, quinolones, tetracyclines, or trimethoprim/sulfamethoxazole. In the event that resistant organisms are likely, the combination of amoxicillin/clavulanic acid or a macrolide such as azithromycin or clarithromycin may be appropriate. Prophylactic antibiotics are not recommended in the long-term care of COPD patients unless other active chronic disease processes such as bronchiectasis or immunodeficiency are present; in such cases, courses of one drug for 2 to 3 weeks followed by another antibiotic for a few weeks may be justified. Finally, amantadine or rimantadine therapy should be considered in unimmunized COPD patients with suspected acute influenza A,¹⁷ if started within the first 48 hours of infection.

Cor Pulmonale. The development of pulmonary hypertension and cor pulmonale in patients with COPD generally indicates a poor prognosis. The primary etiology for these changes is hypoxemia, with the indications for and outcomes of oxygen therapy previously described. Various vasodilators have been investigated in an attempt to reduce pulmonary hypertension and its sequelae. However, pulmonary vasodilators in general have unclear benefits and can have significant negative impacts on most patients, including systemic hypotension and worsening hypoxemia. One case report demonstrated an improvement in pulmonary artery pressures and pulmonary vascular resistance by use of prazosin, an α -blocking agent.¹⁴⁰ In addition, theophylline has significant effects on the pulmonary vasculature that may persist on a long-term basis.¹⁴¹ Diuretics may improve right and left ventricular function in selected individuals with significant congestive heart failure. In the event that diuretics are utilized, alertness to side effects such as electrolyte imbalance and hypotension is vital. Digoxin is not indicated in the treatment of cor pulmonale unless left-sided congestive heart failure is also present.¹⁴² Digoxin is useful in rate control for chronic atrial fibrillation.

Respiratory Stimulants. Various respiratory stimulants, including medroxyprogesterone, acetazolamide, and doxapram, have been suggested as a means of increasing ventilation, reducing hypercapnia, and improving oxygenation in COPD patients. Unfortunately, the stimulating effects of these agents are frequently shortlived, the medications can have side effects, and stimulation of overworked respiratory muscles may induce respiratory muscle fatigue and worsening respiratory failure. Although a European drug, almitrine, is of some value in selected patients, respiratory stimulants are not currently recommended in the United States for hypercapnic or hypoxemic COPD patients. In selected cases, noninvasive venti-

latory support may be utilized in the treatment of acute and chronic respiratory insufficiency, with reduced morbidity and mortality during acute exacerbations and improved exercise tolerance and dyspnea with long-term use.¹⁴³

Dyspnea. On occasion, opiates may be of benefit in severely dyspneic patients. Treatment with morphine and dihydrocodeine can increase exercise tolerance and reduce dyspnea in selected patients; perhaps oral dosing or a skin patch is justified in such individuals.¹⁴⁴ Little is known about the long-term effects of treating dyspnea with opiates and caution is necessary due to the risk of respiratory depression. Benzodiazepines have been shown to have no direct effect on dyspnea. Phenothiazines such as prochlorperazine and promethazine may have some benefit in the treatment of dyspnea, although their impact is small. The appropriate use of psychoactive agents, including anxiolytics and antidepressants, can be of help to COPD patients when severe anxiety or depression is present.

Antiproteases and Antioxidants. Recognition of α -1-antitrypsin deficiency as a cause for the early development of emphysema has led to an increasing awareness of the role of protease/antiprotease and oxidant/antioxidant imbalances in the development of COPD.¹⁴⁵ IV (intravenous) replacement therapy is able to produce appropriate levels of α -1-protease inhibitor in the blood and alveolar fluid.^{146,147} However, inhaled therapy has not been demonstrated to sufficiently penetrate into the lung parenchyma, the likely site of damage in deficient patients. Interestingly, cigarette smoke is capable of impairing normal α -1-protease inhibitor function by altering key molecular sites in the antiprotease protein. Although accepted therapy, the long-term benefits of α -1-antiprotease replacement therapy in α -1-antiprotease-deficient patients remains unproven.

In addition to α -1-antitrypsin deficiency, other protease/antiprotease and oxidant/antioxidant imbalances may contribute to COPD.⁵⁷ New antiprotease and antioxidant agents are at various stages of development. Even nonspecific antioxidants such as vitamins C and E have been proposed as treatments for this disease. However, these agents and dietary manipulation (eg, using fish oil, fresh fruits and green-leaf vegetables, or spicy foods), which may be beneficial, have yet to undergo large-scale clinical trials and remain unproven.

Oxygen

Hypoxemia in COPD patients has been shown to be an indicator of diminished patient function and of mortality; correction of hypoxemia has been documented to improve survival in hypoxemic COPD patients.^{148,149} Most COPD patients with chronic hypoxemia have few if any symptoms, besides dyspnea and exercise limitations, directly attributable to a decrease in oxygenation. Thus, assessment of arterial oxygenation is

necessary in all COPD patients at risk for developing hypoxemia. In addition to its ability to reduce mortality, oxygen therapy for ambulatory outpatients can be expected to achieve early physiologic and symptomatic benefits. Reduction in dyspnea with activity, increased performance of activities of daily living, improved sleep quality, and improved neuropsychiatric function may all be important symptomatic benefits of long-term oxygen therapy.

Although of benefit to hypoxic COPD patients, there is no evidence of improved function or survival with oxygen use in normoxic patients. Because of the expense of long-term oxygen therapy, strict reimbursement guidelines have been instituted, with current recommended indications for oxygen therapy listed in Table 4. Although most studies describe only resting oxygen criteria, oxygen therapy for patients with normal daytime oxygen levels, but with hypoxemia at night or with exercise, has now been shown to improve exercise tolerance and quality of life and may improve survival.^{150,151} A typical oxygen prescription is written for oxygen by nasal cannulas at a flow rate sufficient to produce a $P_{aO_2} \geq 65$ mm Hg or an arterial oxygen saturation $\geq 90\%$ at rest and with activity. Since nocturnal desaturation is common, the flow rate can either be increased by 1 L/min at night or be more precisely adjusted by recording sleep oximetry.

Table 4. Prescribing Criteria for Long-Term Oxygen Therapy*

United States: CMN Form HCFA-484	
1.	$P_{aO_2} < 55$ mm Hg or $S_{aO_2} < 88\%$ (breathing room air)
2.	$P_{aO_2} < 59$ mm Hg and evidence of at least one of the following: pulmonary hypertension (P wave > 3 mm in leads II, III or a VF), cor pulmonale (dependent edema), or erythrocytosis (Hct $> 56\%$)
Annual update of CMN required for patients meeting criteria of 1. Revised CMN required after 3 months for patients meeting criteria of 2.	
*CMN = Certificate of Medical Necessity. This HCFA-484 single sheet must be completed and signed by a physician. S_{aO_2} = arterial oxygen saturation; P_{aO_2} = arterial oxygen pressure; Hct = hematocrit.	

Various oxygen systems are available for the delivery of oxygen to patients. An oxygen concentrator is the least expensive home oxygen device and may be all that is required for homebound patients. However, keeping patients homebound is not desirable and a separate portable system is required. In addition, the concentrator is dependent on electricity and electricity cost is not covered by insurance. A back-up concentrator and a home electricity generator may also be required in rural areas. Compressed gas is widely available and small portable cylinders can provide up to 10 hours of oxygen when low flow rates are used. However, compressed gas is the most expensive method of oxygen delivery when higher flow rates are required. In addition, this requires frequent home deliveries. Liquid oxygen has the advantage of providing a longer-

lasting home supply and the associated portable system is the lightest and lasts the longest. Liquid oxygen is expensive and is not available in many rural areas.

Patient compliance with oxygen therapy and patient activity levels are often reduced because of limited or unreliable delivery times and equipment services associated with the provision of portable oxygen systems. As a result, various methods designed to conserve oxygen have been developed.^{152,153} Nasal cannulas with oxygen reservoirs collect continuously flowing oxygen while the patient exhales, making more oxygen available during inhalation, thereby reducing the needed flow rates and increasing the hours of portable oxygen time. Pulse oxygen devices deliver oxygen only during inspiration, again reducing oxygen flow demands. The instillation of oxygen directly into the trachea using a small transtracheal catheter has been advocated as a means of reducing oxygen needs and reducing ventilatory demands in the COPD patient. In addition, it can eliminate nasal discomfort and eliminate the cosmetic effects of nasal cannulas.¹⁵⁴ However, this requires a minor surgical procedure, regular catheter care by the patients, and close patient monitoring, and it leads to the risk of increased inspissated secretions and catheter tract infection. In general, improved oxygen delivery systems and oxygen conservation devices can encourage better patient compliance, increase ambulatory capabilities, and increase the effectiveness of oxygen therapy.

11. NLHEP and the Primary Health Provider

Following a planning meeting and a major workshop, a new national strategy aimed at the early identification of and intervention in COPD and related disorders became formalized.^{155,156} The NLHEP is conducted through an Executive Committee with membership from the ACCP, ATS, American College of Physicians, American Association for Respiratory Care, the American Association for Cardiovascular and Pulmonary Rehabilitation, the Society for General Internal Medicine, the National Heart, Lung, and Blood Institute, the National Cancer Institute, and the National Institute of Occupational Safety and Health. The consortium provides powerful support for our efforts in providing primary care education and public education. "Test Your Lungs, Know Your Numbers" (FEV₁ and FVC) is the call to arms for physicians and the public.

With modern training in COPD, it is our premise that the primary care physician and other front-line health care providers should be able to diagnose COPD and treat the great majority of patients with COPD throughout the course of their illness. However, there are several clinical circumstances in which consultation with a pulmonary spe-

cialist is indicated. Patients with severe disease accompanied by complications might best be treated in conjunction with a consultant.

12. Indications for Consultation With a Pulmonologist

1. Severe disease, including:
 - a. persistent dyspnea with activities of daily living despite adequate therapy, or
 - b. frequent recurrent exacerbations of COPD.
 Consultation on these patients may address:
 - a. the evaluation of other etiologies of dyspnea,
 - b. review of the therapeutic regimen and recommendations for revisions or additional therapy,
 - c. consideration of additional therapy for intractable dyspnea, including possible use of narcotics (to partially blunt excessive respiratory drives, especially for subsequent severe exacerbations with acute respiratory failure); also discussion of withholding and/or withdrawing life support measures.
2. Evaluation for an initiation of maintenance oxygen therapy. This includes consideration of nocturnal oxygen therapy for nocturnal hypoxemia and consideration of transtracheal oxygen therapy.
3. Failure to successfully taper the patient from systemic corticosteroids.
4. Consideration of and preoperative assessment for thoracic surgery and other surgery placing the patient at high risk for pulmonary complications.
5. Failure to respond after two sequential courses of antibiotics for an acute exacerbation.
6. Presence of severe purulent chronic bronchitis or bronchiectasis for consideration of long-term intermittent or continuous antibiotic therapy.
7. Persistent pulmonary infiltrate(s) on chest radiograph unresponsive to a course of antibiotics. This includes evaluation for tuberculosis, atypical mycobacterial disease, fungal disease, and lung cancer.
8. Evaluation of sleep disturbances, including suspected obstructive sleep apnea.
9. Management of severe acute respiratory failure. This is particularly indicated if treatment with mechanical ventilation is a consideration.
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Medical Direction of Respiratory Care: Past and Present

James K Stoller MD

Introduction

**History of Medical Direction of Respiratory Care
Current Roles and Responsibilities of the Medical
Director of Respiratory Care and Acute Care
Medical Director as Advocate for Respiratory
Care Practitioners**

Introduction

Since the inception of respiratory care as a profession dedicated to serving patients with cardiopulmonary diseases, respiratory care practice and medical direction have been tightly intertwined. Here, I review the role of medical direction in respiratory care, tracing first important historical milestones in the evolution of medical directorship, then, reviewing the roles and responsibilities of current medical directors of respiratory care, and, finally, the role of a medical director as an advocate for respiratory care as presented through official position papers by professional medical societies.

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The History of Medical Direction of Respiratory Care

The current commemoration of 50 years of organized respiratory care practice in the United States recognizes the incorporation of the Inhalation Therapy Association (ITA) in Chicago on April 15, 1947 as the beginning of organized respiratory care in the United States.¹ Even in these roots of respiratory care, collaborating physicians were involved as members of the original ITA group. As another example of collaboration of physicians and respiratory care practitioners regarding education, the ITA and the American College of Chest Physicians (ACCP) co-sponsored an educational program in 1951. With the 1969 publication of the first edition of "Fundamentals of Inhalation Therapy" by Dr Donald F Egan,² the role of the medical director was first delineated. Specifically, Egan noted that the "medical director must be a physician who is interested in chest diseases and who has had at least some clinical experience or training in this field." He suggested that ideal candidates for medical directorship would be from departments of either anesthesiology or internal medicine given the "common foundation of pulmonary physiology underlying both anesthesiology and inhalation therapy." Recognizing the then embryonic nature of the field, Egan pointed out a "shortage of clinicians able or willing to undertake the supervision of inhalation therapy departments, but

there is a hope for an improved future supply as greater emphasis is placed upon postdoctoral training in chest disease." Also, Egan advocated affording the medical director "considerable authority in establishing the professional policies and practices of inhalation therapy in the hospital although any major policy involving patient care or relationship between the department and staff physician should be approved by the hospital medical board. Such a move would ensure understanding of the policy and guarantee maximum cooperation at all levels." Finally, Egan's early description outlined these responsibilities of the medical director:

1. Provision or supervision of the medical care of patients with respiratory diseases, including consultation and referral, acute respiratory care, ambulatory care, and pulmonary function evaluation
2. Establishment of departmental clinical policies and procedures
3. Supervision of inhalation therapy school and/or in-service training and education
4. Education of medical and nursing staffs
5. Selection and promotion of students, trainees, and therapists
6. Maintenance of personnel and medical records
7. Preparation of the departmental budget.

Even in this early vision, the need for close collaboration between medical director and members of the respiratory care staff, the importance of fluid interaction with hospital policymakers, and the educational and service imperatives of the position of medical director were appreciated with great foresight.

In 1973, 4 years after the publication of "Fundamentals of Inhalation Therapy," national standards for medical directorship in acute care hospitals were first introduced by the Joint Commission on the Accreditation of Hospitals,³ the predecessor of our JCAHO (Joint Commission on the Accreditation of Healthcare Organizations). This early statement carried weight because it established medical directorship as essential for hospital accreditation. Specifically, "there must be a director of respiratory care services who is a doctor of medicine or osteopathy with the knowledge, experience, and capabilities to supervise and administer this service properly. The director may serve on either a full-time or part-time basis." Additional criteria for hospital accreditation were that there "must be adequate numbers of respiratory therapists, respiratory therapy technicians, and other personnel who meet the qualifications specified by the medical staff, consistent with state law." This requirement for medical directorship in JCAHO standards persisted until 1996, with current efforts underway by professional societies to reinstate medical directorship for requirements in the most recent JCAHO standards.

The identity and importance of medical direction of respiratory care has further advanced with the formation of a society, the National Association of Medical Directors of Respiratory Care (NAMDRC) in 1977, dedicated to this role. Formed as an independent organization with 93 original members, the official name of NAMDRC was changed in 1995 to the National Association for Medical Direction of Respiratory Care and has grown to a current membership exceeding 630. The NAMDRC Articles of Incorporation specify the organization's purposes:

1. to act as a national, regional, or local information clearinghouse on matters relating to medical direction;
2. to foster continuing cooperation with related health professionals, eg, respiratory therapists, pulmonary function technicians, and respiratory nurse specialists;
3. to provide information on management technology to the membership;
4. to do appropriate professional studies; and
5. to perform any other functions which will foster the highest quality in standards of respiratory care delivery.

Examples of NAMDRC's efforts and advocacy for respiratory care include efforts to reinstate the JCAHO criteria for medical directorship, efforts to simplify and improve the provision of oxygen to oxygen-requiring air travelers, and efforts to ensure that changing reimbursement legislation does not curtail the availability of supplemental home oxygen for hypoxemic individuals.

Broad-based professional support for collaboration between medical directors and the official respiratory care organization, then the American Association of Inhalation Therapists, led to the formation of the Board of Medical Advisors (BOMA) in 1978.¹ This committee maintains an active role in the current AARC and is comprised of physician representatives of 6 "parent" professional organizations whose members interact with respiratory care practitioners, namely the American Thoracic Society (ATS), the ACCP, the American Society of Anesthesiologists (ASA), the American College of Asthma, Allergy, and Immunology (ACAAI), and the American Academy of Pediatrics (AAP). The charges and roles of BOMA are to advise and consult on medical and educational issues and to approve all matters related to medical issues. The BOMA chair is currently designated to serve as a voting member of the AARC Executive Committee and a non-voting member of the Board of Directors. Furthermore, BOMA is charged with advising and interacting with the medical advisors of the chartered affiliates of the AARC. Close interaction between the AARC Board of Directors and the BOMA chairman preserves the interface between respiratory care practitioners and medical directors that has existed since the inception of the AARC.

In the context of the increasingly important role of medical directorship, the issuance of position statements by official professional medical societies has established medical directorship as a defined and essential component of a health care facility's respiratory care service.^{4,6} One of the earliest of such statements was issued jointly by the ACCP, NAM-DRC, and the AARC in 1986⁴ and articulates the characteristics of a qualified medical director of respiratory care as:

The medical director of any inpatient or outpatient respiratory care service, department or home care agency shall be a licensed physician who has a special interest in knowledge in the diagnosis and treatment of respiratory problems. Whenever possible, the medical director should be qualified in special training and/or experience in the management of acute and chronic respiratory disorders. The physician should be responsible for the quality, safety, and appropriateness of the respiratory care services provided, and require the respiratory care is ordered by a physician with medical responsibility for the patient. The medical director should be readily accessible to the respiratory care practitioners and should assure their competency.

Efforts are currently underway by the ACCP to revisit and possibly to revise this statement to reflect newer aspects of medical direction.*

Two years after this joint statement, the ATS also issued a position paper clarifying the characteristics and responsibilities of a medical director of respiratory care.⁵ Reminiscent of the earlier statements, the ATS statement defined the medical director as a licensed physician with special interest and knowledge of respiratory disorders who, whenever possible, was qualified by special training and/or experience. As also noted in prior physician statements, the medical director should be readily accessible to respiratory care practitioners, should assure their competence, and is responsible for the quality, safety, and appropriateness of respiratory care delivered. The ATS statement further specified the responsibilities of the medical director as assuring competence of the respiratory care practitioners and pulmonary technologists, formulating written policies that govern diagnostic and therapeutic procedures, monitoring quality, safety, and appropriateness, and, in conjunction with suitable hospital committees, reviewing the appropriateness of physicians' respiratory care ordering. Furthermore, the medical director has the charge of participating and developing, evaluating, and introducing new respiratory therapy services, equipment and procedures, and monitoring current services for continued usefulness and cost effectiveness. Finally, the medical director

shares responsibility and provides medical expertise to the technical director regarding equipment, personnel, supplies, budget, space, infection control, policies and procedures, safety, preventive maintenance, medical gas systems, fiscal and regulatory agencies, disaster readiness, and quality assurance. Other responsibilities of the medical director are to obtain expert knowledge about respiratory care, to provide continuing education to respiratory care practitioners, physicians and other health care providers regarding respiratory care, to provide liaison with other allied health care providers regarding respiratory care, and to assure compliance with state, federal, and JCAHO regulations.

Between 1988 and the current writing, the role of respiratory care has grown and expanded to newer health care venues, such as skilled nursing facilities, outpatient settings, day care, and hospices. Formal courses on medical directorship of respiratory care sponsored by the AARC and the dedication of the 13th New Horizons Symposium at the 43rd AARC International Respiratory Congress to the topic, "Interaction and Collaboration between Respiratory Care Practitioners and Medical Directors in Delivering Respiratory Care," serve to emphasize the importance of this role and the continued close intertwining of professional roles with respiratory care practitioners.

Current Roles and Responsibilities of the Medical Director of Respiratory & Acute Care

Having evolved over time since 1947, the current roles and responsibilities of the medical director in the acute care hospital can be discussed in 4 arenas:¹ (1) administrative and supervisory, (2) educational, (3) clinical, and (4) investigational. Also, it should be noted that the recent expansion of health care to nontraditional settings outside the acute care hospital has created new roles and opportunities for medical directors which are beyond the scope of this paper.

As presented in Figure 1, administrative responsibilities of the medical director include developing policies, ensuring that the delivery of services is in keeping with state-of-the-art care (eg, protocols,^{7,9} monitoring appropriateness of respiratory care services and managing misallocation,^{10,11} participating in personnel decisions and assessments, and budget preparation). Supervisory activities include certifying competence of respiratory care practitioners in specialized procedures (eg, arterial line placement, intubation) and observing individual practitioners' day-to-day competence.

Figure 1 also presents educational and clinical responsibilities of the medical director. Fostering continuing respiratory care education is a key responsibility of the medical director, both because maintaining currency is essential for optimal practice and because ongoing licensure requires certified continuing education credits in many states. Teaching by example is a desirable attribute of the medical director who should also provide didactic and bedside instruction.

*Paul Selecky MD, American College of Chest Physicians representative to AARC Board of Medical Advisors, 1997; personal communication.

Responsibilities of the Medical Director of Respiratory Care Services

1. Administrative/supervisory

- a. Develops and approves all departmental policies and procedures
- b. Institutes new respiratory care procedures, including respiratory care protocols
- c. Participates in the selection of new employees and, when necessary, the dismissal of old employees
- d. Participates in budget development and review
- e. Participates in the selection and purchase of new equipment
- f. Oversees quality assurance activities, including monitoring for misallocation of respiratory care services
- g. Remains available to staff for consultation at all times
- h. Maintains knowledge of everyday departmental function, including therapists' performance
- i. Certifies respiratory care personnel for specialized procedures (eg, arterial line placement, intubation)

2. Clinical

- a. Remains available to provide consultation to medical colleagues on matters pertaining to respiratory care
- b. Seeks to attain a reputation among medical colleagues of a capable, caring consultant
- c. May oversee special respiratory care activities, eg, weaning unit, hyperbaric unit, smoking-cessation program, pulmonary function laboratory, pulmonary rehabilitation program
- d. Seeks opportunities for respiratory care for which respiratory care practitioners are the best-suited health care providers
- e. Serves as an advocate for respiratory care practitioners

3. Educational

- a. Personally teaches, both through didactic lectures to respiratory care practitioners and at the bedside
- b. Oversees and helps coordinate educational activities of the respiratory care department, eg, seminars, courses
- c. Helps educate medical colleagues regarding optimal respiratory care practice

4. Research

- a. Personally conducts research regarding areas relevant to respiratory care
- b. Promotes respiratory care research by respiratory care practitioners, eg, by serving as a research mentor or by facilitating the acquisition of research skills

Fig. 1. Responsibilities of the Medical Director of Respiratory Care Services. Adapted from Reference 1.

Another educational responsibility of the medical director is to instruct medical colleagues regarding respiratory care. Mechanisms might include delivering medical grand rounds and other formal didactic lectures and fostering interaction between respiratory care practitioners and medical trainees, particularly regarding clinical practice guidelines and protocols that define optimal respiratory care practice.^{7,8,12} In this regard, several recent studies suggest the value of this educational interaction. For example, in a survey of 41 medical house officers at the Cleveland Clinic Foundation,¹³ responses regarding the educational impact of the respira-

tory therapy consult service (RTCS) showed that 97% of respondents felt that the RTCS enhanced the clinical care of patients and that 56% felt that the RTCS enhanced their knowledge of respiratory care ordering. The other 32% of responding house officers felt that the protocol service detracted from their education regarding respiratory care, and 12% indicated neither effect. Similar preliminary data by Messenger¹⁴ show that physicians often indicated that the use of respiratory care protocols enhanced the timeliness (48.1% of respondents), quality (50%), appropriateness (56%), and the professionalism (41%) of respiratory care.

Non-Physician Providers of Respiratory Care Services.

In the changing health care system that is currently in progress, National Association of Medical Directors of Respiratory Care (NAMDRC) unequivocally supports the premise that respiratory care practitioners are the nonphysician caregivers who are best qualified by both education and examination to render respiratory care services in the hospital and at alternate sites, including the home. Due to the complex nature of these services and the patient risks involved, respiratory care services should be provided under the direction of a qualified medical director. NAMDRC has confidence that outcome studies, which are currently in progress, will provide further scientific validation of the benefits attributable to respiratory care practitioners.

The hours of education and the curriculum required for credentialing of a respiratory care practitioner should be the standard for all nonphysician providers of respiratory care services. Verification of the knowledge and skills acquired through this educational process should be documented by appropriate testing, which includes input from physicians who specialize in respiratory medicine in the preparation of certifying examinations.

The current educational process required for credentialing of respiratory care practitioners makes them best qualified to carry out orders for respiratory care clinical interventions. Therefore, respiratory care practitioners play a critical and unique role in the coordination and utilization of respiratory care services at all sites, which is essential for appropriate patient care.

Fig. 2: National Association of Medical Directors of Respiratory Care Statement: Non-Physician Providers of Respiratory Care Services. From Reference 15, with permission.

Another educational aspect of the medical director's role is to interact and advise local respiratory care training institutions regarding curriculum, scope of practice. On a larger scale, physicians with interest in respiratory care may also provide input regarding respiratory care education through service on official educational committees and boards, such as the National Board for Respiratory Care (NBRC), the Committee on Accreditation in Respiratory Care (CoARC), or BOMA.

Clinical availability and credibility are key attributes of an effective medical director. The medical director must attain the reputation among colleagues of being knowledgeable about the spectrum of pulmonary and critical care issues that involve respiratory care services and should be able and keen to assist respiratory care practitioners and physicians alike in clinical management. With knowledge about the spectrum of clinical services to which trained respiratory care practitioners can contribute, the medical director seeks clinical opportunities for which RCPs may provide the best clinical service among allied health providers and advocate for RCPs' involvement in these roles.

Finally, promoting respiratory care research is another desirable attribute of the medical director. Optimally, the medical director would personally conduct research, promote respiratory care research by others, and serve as a research mentor for respiratory care practitioners eager to learn research methods.

Medical Director as Advocate for Respiratory Care Practitioners

As evidence has mounted demonstrating the efficacy and value of respiratory care practitioners in enhancing allocation of respiratory care and clinical outcomes,⁸⁻¹⁰ various official medical societies have published statements advocating respiratory care practitioners as the preferred health care providers of respiratory care services.¹⁵⁻¹⁸ Specifically, physician statements have been issued by NAMDRC,¹⁵ the ACCP,¹⁶ the ASA,¹⁷ and, most recently, by the California Thoracic Society.¹⁸ As presented in Figures 2 through 5, each of these statements endorses the role of respiratory care practitioners and identifies RCPs as the preferred health care providers of respiratory care services, especially to sicker patients and/or those requiring more technically sophisticated treatment (eg, mechanical ventilation, high-flow oxygen). The premises upon which this advocacy are based include: (1) the special training and expertise accrued by respiratory care practitioners to achieve graduation, (2) satisfaction by RCPs of specific credentialing, licensing, and ongoing educational requirements in order to maintain active clinical practice, and (3) ongoing and close collaboration between respiratory care practitioners and medical directors. Implicit in each of these statements is the view that interaction and collaboration between medical directors and RCPs can enhance clinical outcomes for patients with acute and chronic respiratory care needs.

Role of Respiratory Care Practitioners in the Delivery of Respiratory Care Services

In today's ever-changing health care field, efforts have been made to decrease costs by having a variety of health care providers deliver respiratory care services. We are concerned that the quality of these services may be inferior if the health care provider has not had adequate training and experience. Respiratory care practitioners (RCPs) are particularly qualified to assess patients with respiratory problems and to deliver the various modalities of respiratory care because of their unique educational background and training. Their profession has assured practitioner competence by requiring national accreditation of all training programs. This is supplemented by a national credentialing mechanism, often linked with state licensure. Continued competence is bolstered in almost every state by the legal requirement for continuing education in respiratory care. Further, RCPs provide these services under the direction of a qualified medical director.

Because RCPs have specialized training and experience, they play a vital role in the coordination and utilization of respiratory care services. This role is particularly pertinent in this era of managed care, which has resulted in an increased severity of illness in hospitalized patients, as well as in those cared for in their homes and other out-of-hospital sites. Although other health care providers may possess the necessary training and experience to deliver simple modalities of respiratory care, the RCP is uniquely qualified to assist the physician in assessing the overall respiratory needs of patients, and in recommending and delivering the necessary care. Respiratory care modalities can be most beneficial and cost-effective when the RCP functions within the guidelines of physician-approved respiratory care protocols.

In order to assure the safety, quality and appropriateness of respiratory care services delivered to the patients in need, the American College of Chest Physicians strongly endorses the essential role of the competent RCP in providing respiratory care under the direction of a qualified medical director.

Fig. 3. American College of Chest Physicians Section on Respiratory Care: Role of Respiratory Care Practitioners in the Delivery of Respiratory Care Services. From Reference 16, with permission.

Statement of Support for Respiratory Care Practitioners

Health care organizations have sought to implement the use of substitute caregivers. The American Society of Anesthesiologists (ASA) is particularly concerned about this trend in the area of respiratory care.

Respiratory care is a highly specialized allied health profession. Respiratory care practitioners (RCPs) are trained to care for patients under the supervision of a qualified medical director in multiple clinical settings including home care, subacute care, and hospitalized patients. The patients under their care frequently include a disproportionately sicker population than is the case for most other allied health practitioners and RCPs have responsibility for the control of life support equipment in critically-ill patients. RCPs also play an indispensable role in the coordination and utilization of respiratory care services in these multiple settings.

RCPs undergo unique and rigorous formalized training, the programs of which are nationally accredited. They are qualified by a valid and reliable national testing system. They work under the leadership and guidance of a qualified medical director and have done so for many years.

ASA is deeply concerned about the use of other practitioners' delivering respiratory care services. The standard of care to patients could be compromised unless these other individuals received the same extensive education, training, and competency testing as required of RCPs.

ASA strongly supports the continued use of nationally credentialed respiratory care practitioners working under the supervision of a qualified medical director, as they are the most highly qualified health care personnel to deliver respiratory care services to patients.

Fig. 4. American Society of Anesthesiologists. Statement of Support for Respiratory Care Practitioners. From Reference 17, with permission.

Respiratory Care at Alternate (Out of Hospital) Sites.

Introduction

With the changes that are occurring in the health care delivery system increasingly complex care is being delivered to more severely ill patients in settings other than the acute care hospital. These sites include transitional care units, skilled nursing facilities, nursing homes and the patient's home. The ancillary personnel delivering these services have varying degrees of training. The California Thoracic Society is concerned that some of these personnel are being asked to assess and deliver respiratory care to patients who have problems and needs that the personnel have not been adequately trained to address, presenting a potential risk to the patient. This statement addresses those concerns.

Role of Respiratory Care Practitioners

Respiratory care practitioners (RCPs) are qualified to assess patients with respiratory problems and to deliver the modalities of respiratory care because of their educational background and training. Although other health care providers may possess the necessary training and experience to deliver simple modalities of respiratory care, the RCP is qualified to assist the physician in assessing the overall needs of patients and recommending and delivering necessary care. This care can be both beneficial to the patient and cost-effective, particularly when done within guidelines of physician-approved respiratory care protocols.

The patients under the care of the RCP are frequently a sicker population than are generally cared for by most other allied health

care practitioners. RCPs have responsibility for the control of artificial life support equipment and for the assessment of patients with compromised respiratory status. While many of these patients may appear stable, their lack of pulmonary reserve places them in constant jeopardy. The training and the experience of RCPs ensure appropriate care of this fragile patient group.

Conclusions & Recommendations

1. Respiratory care practitioners are trained to render respiratory care services both in the hospital and at alternate (out of hospital) sites.
2. Respiratory care practitioners should function under the direction of a qualified medical director both in the hospital and at alternate sites.*
3. Simple respiratory care services sometimes can be administered by other health care practitioners, but these practitioners should be held to the same standards of practice that apply to RCPs.
4. Complex respiratory services should be administered by or under the supervision of RCPs. Any alternative personnel delivering such care should be subject to:
 - a. verification of their education and training,
 - b. demonstration of their knowledge and skills by appropriate testing of competency,
 - c. licensure under their scope of practice to deliver such care.

*Refer to California Thoracic Society Position Statement, "Medical Director of Respiratory Care Services and Pulmonary Function Laboratories;" 1996.

Fig. 5. California Thoracic Society Position Paper. Respiratory Care at Alternate (Out of Hospital) Sites. From Reference 18, with permission.

In summary, the medical director maintains an important current role in providing respiratory care services. The close alliance and collaboration between respiratory care practitioners and medical directors has been a defining feature since the inception of organized respiratory care in the United States and remains important today to optimize providing respiratory care.

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Outcome-Based Comparisons Improve Respiratory Care Treatments

I just received my December issue of *RESPIRATORY CARE* and read the article by Hoffman and Smithline¹ that compares the Circulaire[™] to conventional small volume nebulizers (SVN) in the treatment of bronchospasm. I liked the fact that the study was outcome-based using a simple device that should be readily available in every emergency department (ED).

The article also served to remind clinicians that if you get the drug in the lungs (where it will do some good) in greater quantity, a greater patient response should be realized. Additionally the patient will benefit more quickly if this can be accomplished in a shorter time frame. Throughout my 20 years in this industry, this theme has been repeated in a variety of formats including, but not limited to:

1. Comparisons of various brands of nebulizers.
2. Comparisons of continuous versus intermittent nebulization.
3. Comparisons of different drugs and combinations of drugs.
4. Comparisons of metered dose inhalers (MDIs) with SVNs.

5. Comparisons of MDIs with and without spacing devices and holding chambers.
6. Comparisons of different kinds of spacers and holding chambers.
7. Various dosing strategies with SVNs or MDIs.
8. Comparisons of concentrated albuterol (5%) with 20 tidal breaths or 4 vital capacity breaths with standard dosing strategies.

The purpose for these kinds of articles is to stress the importance of getting a sufficient quantity of an effective bronchodilating agent into the lungs, quickly and inexpensively.

In the ED, I have often been approved to double or triple (or more) the standard dose of bronchodilator via whatever SVN that happens to be available to me. We do not usually have the opportunity to evaluate the patient, then choose the bronchodilator delivery method and system most likely to benefit the patient on our initial treatment. For me, it has been much easier and less expensive to evaluate the patient, get the SVN, and very unscientifically provide the patient with a more concentrated bronchodilator solution than the standard dose.

It seems reasonable that if the patient gets the drug into the lungs and the response is

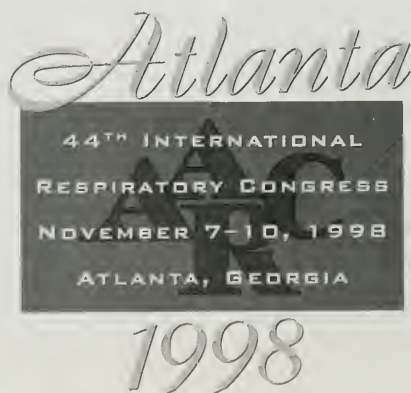
good, we have succeeded in helping the patient, which is our main goal. Being cost conscious, I believe that if I were to give a second or third dose of albuterol out of a multidose vial to a patient in respiratory distress, I would accomplish much more than by using a more expensive device with standard dosing regimens. I fully realize that this should only be done after consulting with the ED physician or another physician knowledgeable in such things, but I have found this approach to be quick, easy, inexpensive, and effective.

I further believe that well-trained, experienced clinicians should assert themselves with the physician staff at their respective institutions to use these more rational (or radical) approaches to helping the patients we serve.

Bruce G Hayton BA RRT
Post Falls, Idaho

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c a l l f o r a b s t r a c t s

1998 RESPIRATORY CARE OPEN FORUM

The American Association for Respiratory Care and its science journal, *RESPIRATORY CARE*, invite submission of brief abstracts related to any aspect of cardiorespiratory care. The abstracts will be reviewed, and selected authors will be invited to present posters at the OPEN FORUM during the AARC International Respiratory Congress in Atlanta, Georgia, November 7–10, 1998. Accepted abstracts will be published in the October 1998 issue of *RESPIRATORY CARE*. Membership in the AARC is not required for participation. All accepted abstracts are automatically considered for ARCF research grants.

SPECIFICATIONS—READ CAREFULLY!

An abstract may report (1) **an original study**, (2) **the evaluation of a method, device or protocol**, or (3) **a case or case series**. Topics may be aspects of adult acute care, continuing care/rehabilitation, perinatology/pediatrics, cardiopulmonary technology, or health care delivery. The abstract may have been presented previously at a local or regional—but not national—meeting and should not have been published previously in a national journal. The abstract is the only evidence by which the reviewers can decide whether the author should be invited to present a poster at the OPEN FORUM. Therefore, *the abstract must provide all important data, findings, and conclusions*. Give specific information. Do not write general statements, such as “Results will be presented” or “Significance will be discussed.”

ESSENTIAL CONTENT ELEMENTS

Original study. Abstract *must* include (1) Background: statement of research problem, question, or hypothesis; (2) Method: description of research design and conduct in sufficient detail to permit judgment of validity; (3) Results: statement of research findings with quantitative data and statistical analysis; (4) Conclusions: interpretation of the meaning of the results.

Method, device, or protocol evaluation. Abstract *must* include (1) Background: identification of the method, device, or protocol and its intended function; (2) Method: description of the evaluation in sufficient detail to permit judgment of its objectivity and validity; (3) Results: findings of the evaluation; (4) Experience: summary of the author's practical experience or a lack of experience; (5) Conclusions: interpretation of the evaluation and experience. Cost comparisons should be included where possible and appropriate.

Case report. Abstract *must* report a case that is uncommon or of exceptional educational value and must include (1) Introduction: relevant basic information important to understanding the case. (2) Case Summary: patient data and response, details of interventions. (3) Discussion: content should reflect results of literature review. The author(s) should have been actively involved in the case and a case-managing physician must be a co-author or must approve the report.

FORMAT AND TYPING INSTRUCTIONS

Accepted abstracts will be photographed and reduced by 40%; therefore, the size of the original text should be *at least* 10 points. *Abstracts should be 400 words or less and may have 1 clear, concise table or figure.* A font like Helvetica or Geneva makes the clearest reproduction. The first line of the abstract should be the title in all capital letters. Title should explain content. Follow title with names of all authors (including credentials), institution(s), and location; underline presenter's name. Type or electronically print the abstract *single spaced in a single paragraph in the space provided on the abstract blank*. Insert only one letter space between sentences. Text submission on diskette is encouraged but must be accompanied by a hard copy. Identifiers will be masked (blinded) for review. *Data may be submitted in table form, or a simple figure may be included provided it fits within the space allotted. No figure, illustration, or table is to be attached to the abstract form.* Provide all author information requested. A clear photocopy of the abstract form may be used. Standard abbreviations may be employed without explanation; new or infrequently used abbreviations should be spelled out on first use. Any recurring phrase or expression may be abbreviated, if it is first explained. Check the abstract for (1) errors in spelling, grammar, facts, and figures; (2) clarity of language; and (3) conformance to these specifications. An abstract not prepared as requested may not be reviewed. Questions about abstract preparation may be telephoned to the editorial staff of *RESPIRATORY CARE* at (972) 406-4667.

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Article in a journal carrying pagination throughout volume:

Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. *Respir Care* 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

Bunch D. Establishing a national database for home care. *AARC Times* 1991;15(Mar):61,62,64.

Corporate author journal article:

American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. *Respir Care* 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

Reynolds HY. Idiopathic interstitial pulmonary fibrosis. *Chest* 1986; 89(3 Suppl):1395-1435.

Abstract in journal: (Abstract citations are to be avoided. Those more than 3 years old should not be cited.)

Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). *Respir Care* 1990;35(11): 1087-1088.

Editorial in journal:

Enright P. Can we relax during spirometry? (editorial). *Am Rev Respir Dis* 1993;148(2):274.

Editorial with no author given:

Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). *Lancet* 1992;340(8833):1440-1441.

Letter in journal:

Aelony Y. Ethnic norms for pulmonary function tests (letter). *Chest* 1991;99(4):1051.

Paper accepted but not yet published:

Hess D. New therapies for asthma. *Respir Care* (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura; 1990. p. 76-85.

Corporate author book:

American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd ed. Littleton CO: Publishing Sciences Group; 1977.

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Triage unit sequence #

Page _____ of _____

A. Patient information

1. Patient identifier	2. Age at time of event: or _____ Date of birth: _____	3. Sex ☐ female ☐ male	4. Weight _____ lbs or _____ kgs
In confidence			

B. Adverse event or product problem

1. ☐ Adverse event and/or ☐ Product problem (e.g., defects/ malfunctions)	
2. Outcomes attributed to adverse event (check all that apply)	
☐ death (mo/day/yr)	☐ disability
☐ life-threatening	☐ congenital anomaly
☐ hospitalization - initial or prolonged	☐ required intervention to prevent permanent impairment/damage
	☐ other: _____
3. Date of event (mo/day/yr)	4. Date of this report (mo/day/yr)
5. Describe event or problem	

5. Describe event or problem

6. Relevant tests/laboratory data, including dates

7. Other relevant history, including preexisting medical conditions (e.g., allergies, race, pregnancy, smoking and alcohol use, hepatic/renal dysfunction, etc.)

C. Suspect medication(s)

1. Name (give labeled strength & mfr/labeler, if known)		3. Therapy dates (if unknown, give duration from to (or best estimate)
#1	_____	#1
#2	_____	#2
2. Dose, frequency & route used		5. Event abated after use stopped or dose reduced #1 ☐ yes ☐ no ☐ doesn't apply #2 ☐ yes ☐ no ☐ doesn't apply
#1	_____	
#2	_____	8. Event reappeared after reintroduction #1 ☐ yes ☐ no ☐ doesn't apply #2 ☐ yes ☐ no ☐ doesn't apply
4. Diagnosis for use (indication)		
#1	_____	
#2	_____	
6. Lot # (if known)	7. Exp. date (if known)	
#1	#1	
#2	#2	
9. NDC # (for product problems only)		
-		
10. Concomitant medical products and therapy dates (exclude treatment of event)		

D. Suspect medical device

1. Brand name	
2. Type of device	
3. Manufacturer name & address	4. Operator of device ☐ health professional ☐ lay user/patient ☐ other
5. Expiration date (mo/day/yr)	
6. model #	7. If implanted, give date (mo/day/yr)
catalog #	
serial #	8. If explanted, give date (mo/day/yr)
lot #	
other #	
9. Device available for evaluation? (Do not send to FDA) ☐ yes ☐ no ☐ returned to manufacturer on _____ (mo/day/yr)	
10. Concomitant medical products and therapy dates (exclude treatment of event)	

E. Reporter (see confidentiality section on back)

1. Name & address		phone #
2. Health professional? ☐ yes ☐ no	3. Occupation	4. Also reported to ☐ manufacturer ☐ user facility ☐ distributor
5. If you do NOT want your identity disclosed to the manufacturer, place an "X" in this box. ☐		



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- medications (drugs or biologics)
- medical devices (including in-vitro diagnostics)
- special nutritional products (dietary supplements, medical foods, infant formulas)
- other products regulated by FDA

Report **SERIOUS** adverse events. An event is serious when the patient outcome is:

- death
- life-threatening (real risk of dying)
- hospitalization (initial or prolonged)
- disability (significant, persistent or permanent)
- congenital anomaly
- required intervention to prevent permanent impairment or damage

Report even if:

- you're not certain the product caused the event
- you don't have all the details

Report product problems – quality, performance or safety concerns such as:

- suspected contamination
- questionable stability
- defective components
- poor packaging or labeling
- therapeutic failures

How to report:

- just fill in the sections that apply to your report
- use section C for all products except medical devices
- attach additional blank pages if needed
- use a separate form for each patient
- report either to FDA or the manufacturer (or both)

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May 6-8—Tennessee Society

Annual Convention and Exhibition, Navigating Uncharted Waters, at Meadow View Resort and Convention Center, Kingsport, Tennessee.
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May 12-15—Region II for Respiratory Care

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Contact: (800) 691-3046, Mail Box #3, or visit the Region II web site at <http://www.bright.net/~dsibb/reg2rc.htm>.

May 22—University of Texas Health Science Center at San Antonio, Texas Society (Alamo District), & Wilford Hall Medical Center

3rd Annual Riverwalk Respiratory Care Symposium at the San Antonio Marriott Riverwalk Hotel, San Antonio, Texas.
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June 25-27—California Society

30th Annual Convention—Thirty Something: Three Decades of Progress and Change—at the Monterey Marriott Hotel, Monterey, California.
CRCE: 17 hours.
Contact: To register, call (805) 723-1180.

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April 9-10—InfoCare

The Annual Adult Respiratory/Critical Care Symposium, at the Sheraton Fiesta Beach Resort, South Padre Island, Texas.
Contact: InfoCare at (956) 233-5645 or fax (956) 233-1010.

May 2-4—Sleep Seminar

The Spring Sleep Seminar 1998 held at the Arlington Resort Hotel and Spa, Hot Springs, Arkansas. Keynote speaker is William C Dement MD PhD.
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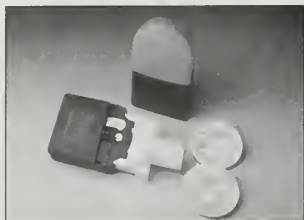
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PEDIATRIC INHALED CORTICOSTEROID. GlaxoWellcome releases Flovent® Rotadisk® for children, ages 4 to 11 years. The first and only, the company claims, anti-inflammatory corticosteroid medication that can be used for preventive asthma therapy in children as young as 4 years. The inhaled powdered corticosteroid is available in doses of 50 and 100 mcg for twice daily use. Details concerning the side effects, indications, and contraindications are available from GlaxoWellcome. Circle Reader Service Number 160.



ENDOTRACHEAL TUBE SECURING DEVICE. TrachTape, a new device for securing endotracheal (ET) tubes is available from Welcon. TrachTape eliminates the need for "makeshift" tube-securing devices, is suitable for use with a variety of patients—even those with facial trauma, and will not absorb water or body fluids, the manufacturer claims. For a free sample from Welcon, circle Reader Service Number 161.

NASAL MASK. Respiration Inc designs the new GoldSeal™ Nasal Mask. The mask is used for the treatment of ob-

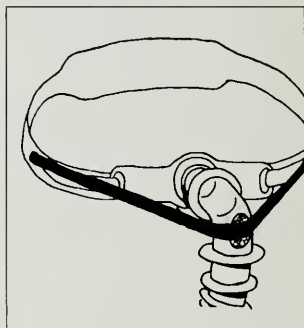
structive sleep apnea, respiratory failure, or respiratory insufficiency and can be used with Respiration CPAP (continuous positive airway pressure) or BiPAP® Systems. The mask is constructed with a gel-like material that allows an effective seal with little headgear tension, a company press release says. In addition, monitoring ports allow pressure to be measured at the mask, a swivel allows the circuit tubing to move with the patient, and an optional support ring or foam spacers add support to the mask. To learn more about the mask that is available in 7 sizes, circle Reader Service Number 162.



PEDIATRIC MOUTHPIECE. SafeTway™, from Vitalograph, is now available in pediatric-size versions. The device reduces the possibility of crossinfection and the need for cleaning pulmonary function testing equipment between patients, according to a Vitalograph press release. In addition, the mouthpiece incorporates a 1-way valve to prevent the patient from inhaling, thereby reducing the chances of the patient's breathing contaminated air. For more information about the device, circle Reader Service Number 163.

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ANTIDISCONNECT DEVICE. Pepper Medical Inc, formerly Zipper Medical Inc, introduces the Zipper Medical Ventilator Antidisconnect Device. The device is an endotracheal tube neckband coupled with a patented antidisconnect strap that helps prevent disconnection from the ventilator. The neckband is made of soft cotton with hook-and-loop fasteners and adjusts to a variety of neck sizes, according to company literature. For more information about the Antidisconnect Device, circle Reader Service Number 165.

OFFICE SUPPLY CATALOG. The 1998 spring/summer Catalog of Office Essentials for Medical Professionals is now available from Medical Arts Press. The 200-page catalog features office supplies, stationery, insurance forms, clinical forms, and over 250 new products. Products for building a practice and establishing patient loyalty are also included. circle Reader Service Number 166 to request your free copy of the catalog.

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For an application, call (800) 867-7001. Completed applications must be received by May 1, 1998. Grants will be awarded in October.

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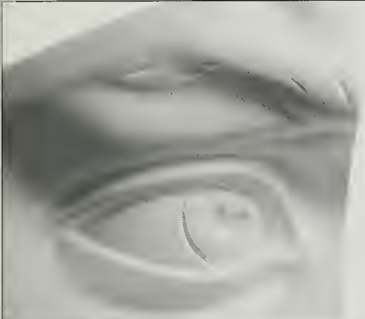
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RRT Examination	June 6, 1998 Application Deadline: February 1, 1998	120 Written only (new applicant) 80 Written only (reapplicant)
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	December 5, 1998 Application Deadline: August 1, 1998	250 Both (new applicant) 210 Both (reapplicant)
CPFT Examination	June 6, 1998 Application Deadline: April 1, 1998	130 (new applicant) 100 (reapplicant)
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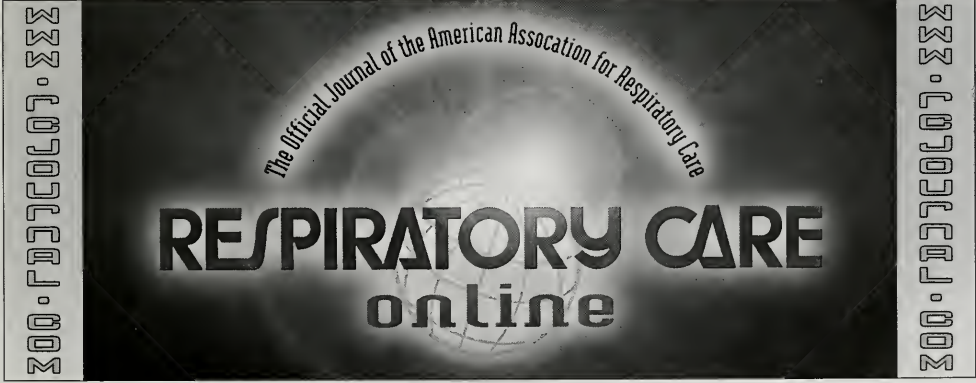
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9. ☐ Rehabilitation
10. ☐ Education

IV. Position

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- B. ☐ Chief Therapist
- C. ☐ Supervisor
- D. ☐ Staff Therapist/ Technician

- E. ☐ Medical Director
- F. ☐ Educator/Instructor
- I. ☐ Sales
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